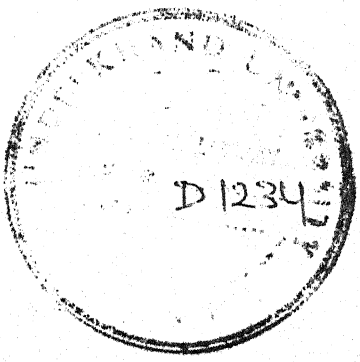
CLINICO-ECHOCARDIOGRAPHIC EVALUATION OF PATIENTS OF ACUTE MYOCARDIAL INFARCTION

THESIS
FOR
Doctor of Medicine
[MEDICINE]



M.L.B. MEDICAL COLLEGE

BUNDELKHAND UNIVERSITY
JHANSI [UP]

2002

DEEPSHIKHA

Certificate

This is to certify that work entitled "Clinico-echocardiographic Evaluation of Patients of Acute Myocardial Infarction" is being submitted as a thesis for M.D. (Medicine) examination, 2002 Bundelkhand University, has been carried out by Dr. Deepshikha in the Department of Medicine, MLB Medical College, Jhansi Bundelkhand University.

She has put in necessary stay in the Department of Medicine as per University regulations.

Dated : 6./3./02

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(Guide)

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Dated : 9../.3../02

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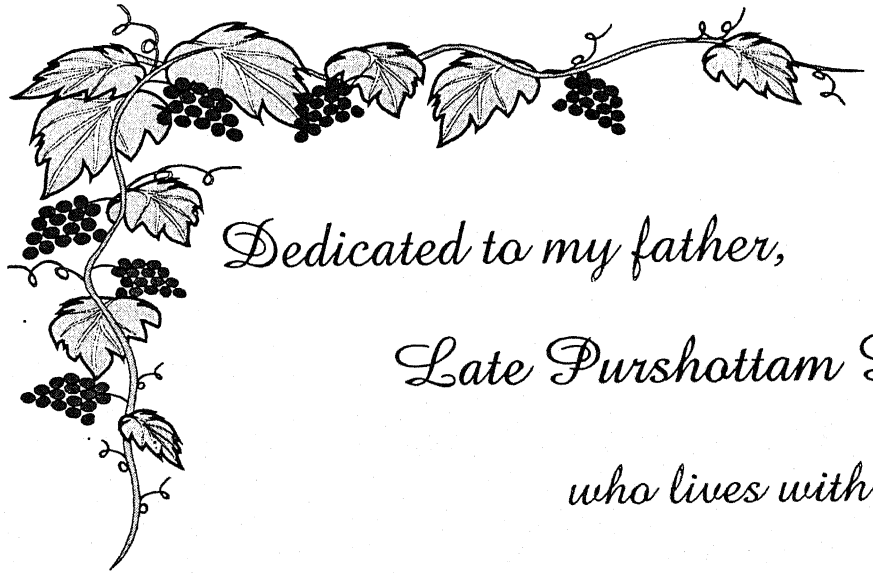

Dedicated to my father,

Late Purshottam Prasad

who lives with me, in me,

every moment ...

Acknowledgment

"Life's battles don't always go
To the stronger or faster man,

But sooner or later the man who wins
Is the man who thinks he can"

When any piece of work is satisfactory accomplished it is never the work of one person but a concerted effort of a number of people who silently work behind the scene and often go unheard. It is to those people I feel indebted and without whose help I could not completed this work. Their knowledge and practical experience have graded me to complete this work.

My first thought is to offer my obeisance to GOD almighty without whose silent but omnipresent help, none of this work have been possible.

This is the virtue that I have observed in our esteemed and able teacher my guide Dr. Praveen Kumar Jain, MD Dip. card. DM (Cardiology) Professor & incharge,Cardiology unit, Department of Medicine, M.L.B. Medical College, Jhansi. The profound work has taken shape only because of his wise counsel, firm initiative, timely constructive criticism, sympathetic attitude and invaluable advice. It was the brilliance of his presence that made it possible for me to traverse the

path of success. He guided me at every point with his invaluable suggestions, meticulous attention and perfused my brain with his unlimited knowledge.

"Take time to work, it is the price of success
Take time to think, it is the source of power
Take time to read, it is the foundation of wisdom"

This is what our honourable professor and Head Dr. R.C. Arora, MD, D.Sc. Department of Medicine, M.L.B Medical College, Jhansi, who is my co-guide has made me inculcate amidst my stay in the Department. His inspiring and considerate nature infuses immense confidence in students, who always acted as a beacon of light to me. He has been a perpetual source of inspiration and knowledge throughout this study. His strong discipline strives to get the best out of every student. His cool and composed self is a source of never ending example and he personifies the rock of gibralter in the Department.

Think big, dare to dream, dare to try
Dare to fall, dare to succeed.

This has been always taught by our teacher Dr. P.K. Jain, MD, MNAMS Professor, Department of Medicine, M.L.B Medical College, Jhansi. I acknowledged with sincere thanks, the heartening words and constant encouragement that has been rendered to me during my stay in the Department. His perpetual zest for knowledge acts as a catalyst to infuse enthusiasm in the students for the same.

I would like to thank Dr. Navneet Agrawal MD, Professor, Dr. N.S. Sengar MD, DM (Nephrology) Lecturer, Dr. Gyanendra Kumar, MD, Lecturer, Department of Medicine, M.L.B Medical College, Jhansi for their encouraging and helpful attitude.

I am highly indebted to my friends and colleagues especially Dr.Deep Chandra Pant, Dr. K.S. Prasanna , Dr. Zaheer Hussain, Dr. Upendra Singh, who provided me every possible help in building up the infrastructure for this work and made completion of this towering job smooth and manageable.

Lastly I pay my sincere most thanks to my mother. And Words fails to express my deep sense of gratitude to my father (Late Purshottam Prasad), who made me realize that I was capable of achieving a goal. He encouraged me to prove my worth. He made me what I'm today. He is the man behind all my success.

The excellent documenting and printing of this work requires special mention for which I'm thankful to Mr. Farhan, Jhansi.

Deepshikha

Dated : (Deepshikha)

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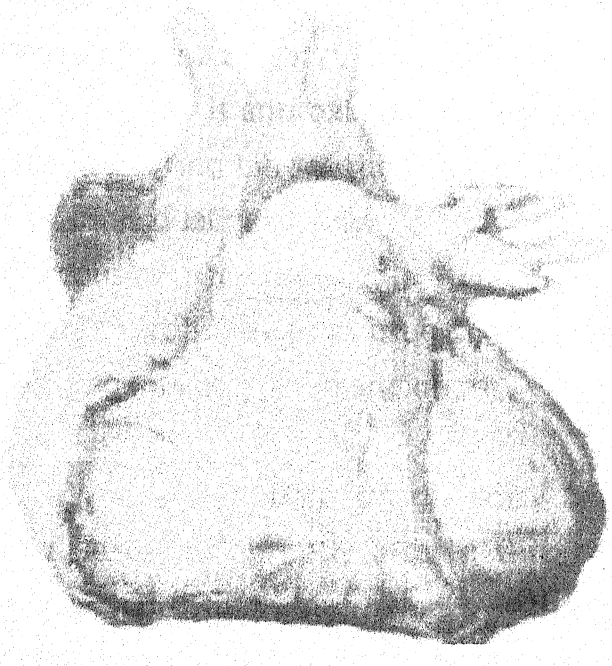


INTRODUCTION

INTRODUCTION

In the United States approximately, 700000 coronary artery related deaths and more than 1.1 million Acute Myocardial Infarctions occur each year[1]. The mortality rates with Acute Myocardial Infarction is approximately 30%, with more than half of these deaths occurring before the patient reaches the hospital. The decreasing individual mortality from myocardial infarction is possibly accounted for by earlier detection, use of intensive coronary care unit, advances in drug therapy and reperfusion with thrombolytic agents or balloon angioplasty.

Acute Myocardial Infarction generally occurs when coronary blood flow decreases abruptly after a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis and which leads to reversible myocardial cell damage and necrosis. Coronary thrombus is formed at the site of vascular injury. This injury is produced by various factors e.g. cigarette smoking, hypertension and hyperlipidemia. In most cases infarction occurs when atherosclerotic plaque fissures, ruptures or ulcerates and when conditions favour thrombogenesis, so that a mural thrombus forms at the site of rupture and leads to coronary artery occlusion. Histologic studies show that the coronary plaques prone to rupture are those with rich lipid core and thin fibrous cap. Such plaques usually have an eccentrically located lipid pool. Plaque rupture usually occurs at the junction between the fibrous cap and the normal vessel wall, probably from stress at this area.

Whether increased shear forces caused by the stenosis, repeated oscillatory stress resulting from contraction of the heart or changes in coronary tone related to circulating catecholamine act single or in concert to potentiate plaque rupture remains conjectural.

After the rupture of plaque, initial platelet monolayer forms and then various agonists e.g. collagens ADP, epinephrine, serotonin etc. promotes platelet activation. After platelet activation there is production and release of thromboxane A2, further platelet activation and potential resistance to thrombolysis.

Activation of platelets by agonists produce a conformational change in the glycoprotein IIb/IIIa receptor. When glycoprotein IIb/IIIa converted to its functional state, this receptor develops high affinity to vWF and fibrinogen. Since vWF and fibrinogen are multivalent molecules they can bind to two different platelets, simultaneously resulting in platelets cross linking or aggregation.

The coagulation cascade is activated on exposure of tissue factor in damaged endothelial cells at the site of ruptured plaque. Factors VII & X an activated, & leading to the conversion of prothrombin to thrombin which then converts fibrinogen to fibrin. Thrombin further leads to auto amplification reaction that results further activation of the coagulation cascade. The athero-sclerosed artery eventually become occluded by a thrombus containing platelet aggregates & fibrin strands.

Infrequently Myocardial Infarction may occur with prolonged or severe coronary spasm in the absence of under lying coronary

artery disease e.g. cocaine use, ergot therapy and severe emotional stress.

Following are rare cause of Myocardial Infarction –

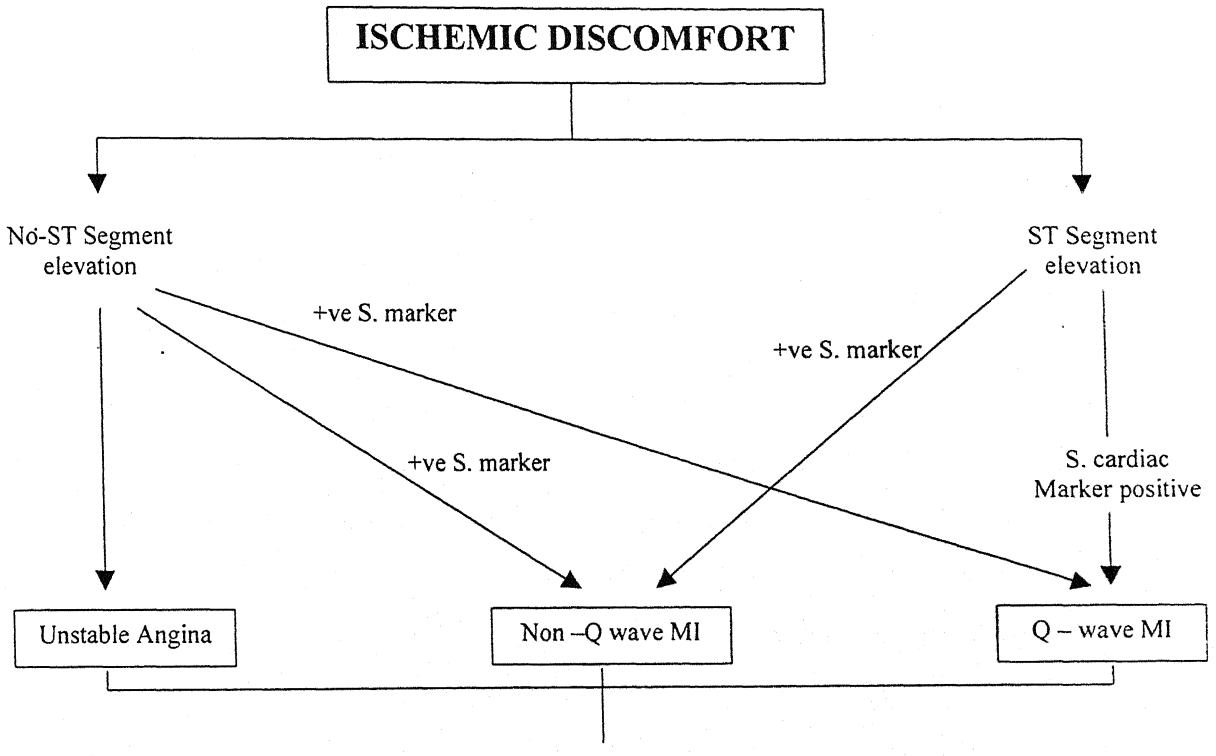
1. Spontaneous coronary artery dissection
2. Serum Sickness
3. Various allergic conditions
4. Profound hypoxemia
5. Sickle cell crisis.
6. Carbon mono-oxide poisoning
7. Acquired hyper-coagulable state

There is an increase in Myocardial Infarction event rates in the early morning hours, probably related to circardian variation in coronary vascular tone, catecholamines and co-agulability.

Totally occlusive thrombus in patients with inadequate distal collateralization most often result in Q-Wave Myocardial Infarction. Transiently occluding thrombus, with spontaneous lysis or distal collateralization may yield lesser degree of necrosis and produce "non-Q" wave Myocardial Infarction.

Patients with intermittent or subtotal occlusive thrombi, adequate collateralization or both may have the syndrome of unstable or prolonged angina in the absence of myonecrosis or if serum cardiac markers are detected as having "non Q" Wave Myocardial Infarction, a minority of patients who present initially without ST segment elevation may develop a "Q" wave myocardial infarction. The presentation that constitute the spectrum ranging from unstable angina through 'non-Q' wave Myocardial Infarction to

'Q' wave Myocardial Infarction are called **Acute coronary syndrome.**[2]



SERUM CARDIAC MARKERS –

1. Creatine – Phospho Kinase MB – appears after 4-6 hrs. and peak level reaches at approximately 20 hrs. after the coronary occlusion and falls to normal within 48-72 hrs.

2. Troponin I & T – These are normally not detected in the blood of healthy individuals but may increase after myocardial infarction to levels over 20 times higher than the cut off value. Trop I may remain elevated for 7 to 10 days after myocardial infarction and Trop T level may remain elevated for upto 10 to 14 days.

3. LDH – It comes in blood more than 24 to 48 hrs after the onset of symptoms and reaches a peak after 2 or 3 days and may elevated for a week or more.

4. AST – It starts to rise about 12 hrs after infarction and reaches a peak on the first or second day, returning to the normal within 3 or 4 days.

MORTALITY :

The mortality rate with Acute Myocardial Infarction is approximately 30% more than half of these death occurring before stricken individual reacher to the hospital. Approximately 1 of every 25 patients who survive the initial hospitalisation, dies in the first year after myocardial infarction. Survival is reduced in over age i.e. > 75yrs., whose mortality rate is 20% at one month and 30% at one year after Acute Myocardial Infarction.

COMPLICATIONS :

Complications of Acute Myocardial Infarction occur in a time dependent manner and can be directly related to the anatomy of the coronary artery blood supply. Early complication may begin within 20min of the onset of Myocardial Infarction. These complications include arrhythmias and heart block (due to injured or ischemic conduction system) and hypotension and congestive heart failure due to ischemic or injured muscle tissue resulting in abnormal filling (diastolic dysfunction) or abnormal emptying (Systolic dysfunction).

Several days later complications of Myocardial Infarction can occur due to "Yellow softening" of myocardial tissue resulting in one

of several "Mechanical complications" of Myocardial Infarction in addition inflammation surrounding the injured myocardial tissue can lead to postinfarction pericarditis.

1. Ventricular Dysfunction :

After myocardial infarction the left ventricle undergoes a series of changes in shape, size and thickness in both the infarcted and non-infarcted segment. This process is known as ventricular remodeling. Soon after Myocardial Infarction the left ventricle begins to dilate resulting in disproportionate thinning and elongation of the infarcted zone, later lengthening of the non-infarcted segment occur as well with greater dilatation following infarction of the apex of the left ventricle & causing more marked hemodynamic impairment and frequent heart failure and poorer prognosis. Degree of pump failure correlates hemodynamic assessment of left ventricular failure well with the extent of ischemic necrosis and with mortality. The diagnostic signs are pulmonary rales, S3 and S4 gallop rhythm and pulmonary congestion on X-ray chest. Hemodynamic evidence of abnormal left ventricular function appears when contraction is seriously impaired in 20% to 25% of the left ventricular wall.

2. Cardiogenic Shock

Typically patients who develop cardiogenic shock have severe multivessel coronary artery disease. Now-a-days the incidence of cardiogenic shock is about 7%. Only 10% of patients with this condition present with it on admission. While 90% develop during hospitalization. This is characterized by marked hypotension i.e. (<90 mm Hg systolic and decrease in cardiac index (approximately <1.8

L/min/mm^2). Cardiogenic shock is generally associated with a mortality rate of 70%[3,4]. Infarction of 40% or more of the left ventricle usually results in cardiogenic shock.

Cardiogenic shock results when there is a marked reduction in forward cardiac out put leading to hypotension, decreased organ perfusion and at the same time elevated left ventricular filling pressure leading to congestive heart failure. This can be due to either massive left ventricular complication e.g. Mitral Regurgitation ventricular aneurysm formation, Right ventricular infarction.

3. Right Ventricular Infarction[5,6] :

Approximately 1/3 of patients with inferioposterior area infarction demonstrate a minor degree of right ventricular necrosis. Clinically significant right ventricular infarct presents with increase JVP, Kussmaul's Sign, hepatomegaly with or without hypotension. ST segment elevation of the V1 and V2 and particularly V4R in the first 24 hours.

Right Ventricular infarction occurs almost exclusively in the setting of Right coronary artery occlusion.

2D echo is helpful in determining the degree of right ventricular dysfunction.

4. Mechanical cases of heart failure : it includes

a) Acute mitral regurgitation.
b) Free wall rupture,
c) Left ventricular aneurysm formation.

d) Ventricular septal defect.

a) *Acute Mitral Regurgitation*

Acute mitral regurgitation may occur abruptly from rupture of a left ventricular papillary muscle resulting in a flail mitral leaflet, usually the posterior leaflet. This result in an abrupt decrease in forward cardiac output, leading to congestive heart failure and often to the cardiogenic shock.

This occurs more commonly in the setting of inferior wall Myocardial Infarction since the Right Coronary Artery and Left circumflex artery supply the posteriomedial head of the papillary muscle, which is more prone to rupture than anterolateral head. Ventricular septal defect and mitral regurgitation are often impossible to be differentiated from each other because both presents with pansystolic murmur and tall 'V' wave in pulmonary capillary wedge pressure. Colour Doppler echocardiography may help in differentiating these two conditions. Unlike rupture of ventricular septal defect which occurs with large infarct, papillary muscle rupture occurs with a relatively small infarction[7].

b) *Left ventricular free wall rupture*

Rupture of the left ventricular free wall is analogous to ventricular septal defect but occurs in the free wall of the left ventricle, usually resulting in abrupt cardiogenic shock due to cardiac tamponade. Rarely a pseudoaneurysm of the left ventricle occurs if there is incomplete rupture of the free wall and this may be undetected clinically until abrupt deterioration occurs.

However, as a group they are probably responsible for about 15% of all deaths from Acute Myocardial Infarction[8,9].

c) *Left ventricular aneurysm formation :*

Left ventricular apical aneurysm formation usually occurs following antero-apical myocardial infarction after Left Anterior Descending occlusion. This weakening of the apical wall results in an out pouching or dyskinesis of the apex of the heart during systole.

The resultant stasis of blood in the dyskinetic segment of the apex may result in thrombus formation and systemic embolization. The reduced left ventricular ejection fraction may lead to congestive heart failure and predispose to ventricular arrhythmias.

It is of 2 type Viz true aneurysm and false aneusysm. Ventricular aneurysm are readily detected by 2 D echo which may also reveal a moral thrombus in an aneurysm. True Left Ventricular aneurysm occurs probably of Acute Myocardial Infarction and are more frequently in patient with transmural myocardial infarction.

d) *Ventricular Septal rupture :*

Acute ventricular septal rupture can occur usually several days following the acute infarction, due to softening of the necrotic tissues of septum. This can occur in both inferoposterior and in anterior Myocardial Infarction. A loud systolic ejection murmur

usually occurs and results in an acute left to right shunt with congestive heart failure and usually cardiogenic shock.

5. ARRHYTHMIAS :

The mechanism responsible for infarction related arrhythmias include autonomic nervous system imbalance, electrolyte disturbance, ischemia and slowed conduction, mortality form arrhythmias is greater during the first few hours.

6. PERICARDITIS :

Pericardial frictions rub and or pericardial pain are encountered in about 10% patient with acute transmural myocardial infarction. Anti co-agulants potentially could cause tamponade, in the presence of acute pericarditis.

Infrequently Dressler's Syndrome[10] or post myocardial infarction syndrom[11] may be present which is characterized by fever and pleuropericardial pain. It may begin from few days to 6 weeks after myocardial infarction.

7. THROMBOEMBOLISM :

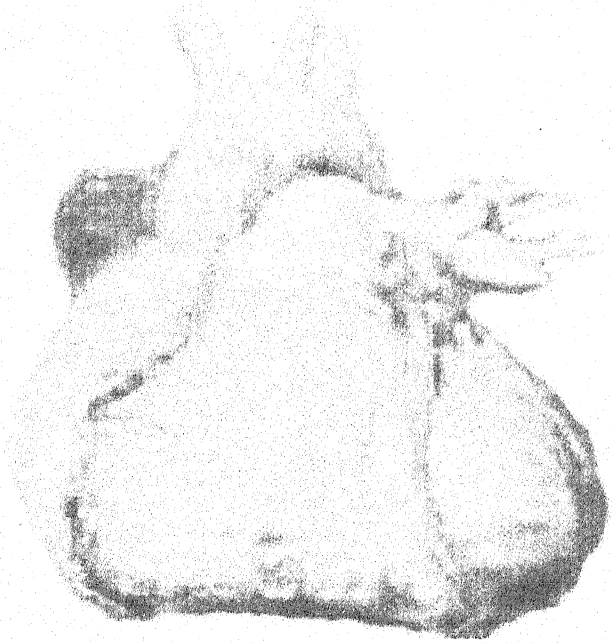
Clinically apparent thromboembolism complicate Acute Myocardial Infarction in approximately 10% of cases but embolic lesions are found in 20% of patients in necropsy series, suggesting that thromboembolism is often clinically silent. Thrombus can be detected by echocardiography. Echocardiography reveals left ventricular thrombi in about 1/3 of the patients with anterior

infarction but also in few patients with inferior or posterior infarction.

8. HYPOTENSION :

Hypotension may occur in various setting following Acute. Myocardial Infarction. These include –

1. Hypovolemia.
2. Excessive vasodilatation from nitrate therapy.
3. Decreased left ventricular filling, secondary to right ventricular infarction.
4. Marked reduction in cardiac output to extensive infarction or to a mechanical complication.



AIMS & OBJECTIVES

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1. To do clinico – echocardiographic evaluation in patients of Acute Myocardial Infarction.

2. To study the various complications in patients of myocardial infarction and their followup.

REVIEW OF LITERATURE

REVIEW OF LITERATRE

Myocardial infarction is almost always due to the formation of occlusive thrombus at the site of rupture of an atheromatous plaque in a coronary artery. In United States approximately 70,0000 coronary artery related deaths and more than 1.1 million Acute Myocardial Infarction occur each year. The epidemics of cardiovascular disease especially coronary heart disease are emerging in India. In 1990 cardio vascular disease accounted for 2.4 million deaths in India with an annual loss of 28.5 million disability adjusted life years[12]. The factors underlying the special predilection of South Asian for coronary heart disease are a high prevalence of glucose intolerance, central obesity, elevated plasma triglycerides, decreased plasma HDL cholesterol & hyper insulinemia. The confluence of genetically determined elevation of lipoprotein 'a' (LP(a)), possible genetic susceptibility to the metabolic syndrome and environmental influence which enhance the propensity for diabetes and elevated total cholesterol level appears to make the South Asian immigrants especially vulnerable to coronary artery disease.

The excess of coronary mortality and morbidity noted in persons of South Asian ethnicity in comparison to many other ethnic groups, implicate gene environment interactions which amplify the risk for Indians. Such susceptibility may be due to a "thrifty gene" selecting for survival over millennia or a thrifty phenotype emerging alive from the adverse intra-uterine environment of an impoverished pregnancy.

Studies of urban rural comparison within Indian and comparison of migrants with non migrants siblings, however, suggest that environment is a major determinant of the extent to which such susceptibility expresses itself as cardio vascular disease whether the programming is due to mother nature or mother's nurture[13,14,15].

VENTRICULAR DYSFUNCTION

Even in the thrombolytic era, left ventricular dysfunction remains the single most important predictor of mortality following Acute Myocardial Infarction. In patients with Acute Myocardial Infarction heart failure is characterised either by systolic dysfunction alone or by both systolic and diastolic dysfunction. Left ventricular diastolic dysfunction leads to pulmonary venous hypertension and pulmonary congestion, whereas systolic dysfunction is principally responsible for a depression of cardiac output and of the ejection fraction, Clinical manifestation of left ventricular failure become more common as the extent of the injury to the left ventricle increase. The dysfunction however is due to 2 components:

(i) Irreversible i.e. due to necrosis of the myocardium.

(ii) Reversible this may be due to stunned or hibernating myocardium.

(a) *Stunned Myocardium*[16] *:* Left Ventricle dysfunction due to the transient coronary occlusion followed by restoration of blood flow even though myocardial necrosis is minimal or absent. This may persist for hours to several weeks.

(b) *Hibernating Myocardium16* : This is chronic depression of left ventricular function caused by prolonged periods of ischemia due to severe obstruction in coronary arteries. This coronary blood flow is inadequate to support myocardial contractile function but is adequate to maintain cellular structure and integrity. If myocardial perfusion is restored, systolic function improves. Duration may vary from months to years.

Diastolic Dysfunction : Implies elevation of left ventricular end diastolic pressure (LVEDP) with normal left ventricle end diastolic Volume (EDV) (55-85 ml/mt) and normal ejection fraction.

Systolic Dysfunction : Implies raised EDV with LVEDP and diminished EF. Systolic dysfunction is more common (70%) than diastolic dysfunction (30%).

* 2 D – Echo Doppler has emerged as the modality of choice for evaluating diastolic & systolic dysfunction.

S.H. Poulser et al[17] had studied 65 consecutive patients admitted to the coronary care unit with first Acute Myocardial Infarction defined according to the WHO criteria with 2 out of the following three characteristics.

1. Typical chest pain.
2. Electrocardiographic evidence of myocardial infarction (ST elevation > 1 mm in contiguous leads or subendocardial injury pattern) and
3. Transient elevation of creatinine kinase MB ≥ 20 U/L (Normal < 6 U/L)

All patients were between 40-75 years of age. No patient had significant valvular heart disease and all were in sinus rhythm. Pulsed Doppler echocardiography of transmitral and pulmonary venous flow was assessed in 65 consecutive patients within 1 hr. of arrival in the coronary care unit. Isolated left ventricular systolic dysfunction (EF < 50% and normal filling pattern) was found in 18 patients (21%). Left ventricular diastolic dysfunction (defined as impaired relaxation or a pseudonormalized or restrictive filling pattern) and a normal systolic function was found in 15 patients (24%). Combined systolic and diastolic dysfunction was found in 24 patients (38%) and combined normal systolic and diastolic function in 11 patients (17%). Left ventricular diastolic dysfunction is present early after onset of symptoms of a first Acute Myocardial Infarction. Further more left ventricular diastolic function seems to play an important role in the development of clinical heart failure following Acute Myocardial Infarction. Impaired relaxation of the left ventricle was the predominant diastolic filling abnormality (37%) but pseudonormal or restrictive filling pattern were also frequently present (25%) in patients with a first Acute Myocardial Infarction. In addition 24% of the patients had isolated diastolic dysfunction. Development of congestive heart failure occurred in 31% of the patients during the first week after the acute events. Although most patients with heart failure had early systolic dysfunction with decreased EF, a sub group (23%) had an abnormal filling pattern with preserved ejection fraction. Patients at risk of developing congestive heart failure during the first week of a first Acute Myocardial Infarction are best identified by a short mitral E deceleration time.

CARDIOGENIC SHOCK[18] :

Cardiogenic shock is defined as a critical reduction in tissue perfusion caused by a critical fall of cardiac output to level that are inadequate to support end organ function. The two cardinal elements of cardiogenic shock are hypotension and hypoperfusion. The clinical presentation of cardiogenic shock includes

1. Systolic pressure < 90mm Hg,
2. Peripheral vasoconstriction with cool extremities and acrocyanosis
3. Urine output – 20 ml/hr
4. Altered mental status with agitation or obtundation

Various pathologic condition in the setting of Acute Myocardial Infarction can induce cardiogenic shock e.g. extensive infarction involving a large area of left/right ventricular myocardium is the most frequent cause, other causes include mechanical complication such as severe mitral regurgitation, ventricular septal defect and cardiac tamponade with or without free wall rupture.

Cardiogenic shock occurs in 6% to 20% of patients with Acute Myocardial Infarction[19,20,21]. In the Worcester heart attack study[22] over a period of 13 years from 1975 to 1988, the incidence of cardiogenic shock ranged from 6.7 to 9.1% in a sample of 4762 patients with Acute Myocardial Infarction admitted to 16 community hospitals. During that time interval, the rate of this complication showed an acute increase after adjustment for base line variables with adjusted relative risk of 0.83 in 1978 & 1.65 in 1988. The in hospital survival of patients enrolled in this study actually worsened between 1975 &

1988 with a mortality rate of 74% in 1975 & of 82% in 1988. Aggressive strategies were applied only infrequently in this study < 2% underwent coronary angioplasty, further more, thrombolytic therapy was infrequently used.

In the Global utilization of streptokinase and Tissue plasminogen activator for occluded coronary arteries **(Gusto-1)** trial of 41,021 patients, cardiogenic shock occurred in 7.2% of patients. The 30 days mortality rate for the patients who had shock was 55% and accounted for 58% of the total mortality of the trial. The comparison between resource consumption in the United States and that of other countries for the patients with shock enrolled in the trial showed that every intervention was performed significantly more frequently in the US patients including cardiac catheterization (58% vs 23%), intra-aortic balloon pump (35% vs 7%), coronary by pass surgery (16% vs 9%) or coronary angioplasty (26% vs 8%). Adjusted 30 days mortality rate was significantly lower among pt treated in United States than among those treated in other countries (50% vs 66%) with a 24% reduction. In the multivariate analysis, age and systolic blood pressure were the only factors more strongly associated with increased 30 days mortality rate than geographical location. In all countries mortality rate at a 1 year was lower for patients who underwent coronary angioplasty, whereas coronary surgery did not significantly affect 1 year mortality.

Bengtson et al[23] showed that the most important independent predictor of in hospital and long term mortality rates was patency of the infarct related artery. The in hospital mortality rate in patients with patent infarct-related arteries was 33% vs 75% in those with closed arteries and 84% in those with unknown status of the arteries. Therefore the main goal of current treatment of

cardiogenic shock in Acute Myocardial Infarction is aggressive myocardial revascularization, aiming to maximize myocardial salvage.

Dzavik et al[24] described significant increase from 1989 to 1995 in the use of cardiac catheterisation interventional procedures and intra-aortic balloon pump in all age groups, although an aggressive strategy was less frequently used in patients > 75 years old. In hospital survival of patients < 65 years old improved from 10% in 1989 to 1990 to 59% in 1993 to 1995, survival of patients > 75 years old was 20% in 1993 to 1995. Use of thrombolytic therapy showed possible survival benefit only in patients > 75 years old (Thrombolysis 33% vs no thrombolysis 5%).

The GISSI study[25] (The Gruppo italianoper to studio della sopravvivenza nell" infarto miocardico) was the only large randomized, placebo-controlled trial of thrombolysis that enrolled patients with shock. In the small cohort including 280 patients, the 30 days mortality rate was 69.9% for patients of the streptokinase group.

In the meta analysis of several large, placebo-controlled, randomized thrombolytic trials by the Fibrinolytic therapy Trialists **(FTT)** collaborative Group[26], the 35 days mortality rate of patients with hypotension (systolic blood pressure < 100 mg hg) treated with thrombolysis was significantly lower that of patients of the placebo group (29% vs 35%). When both hypotension and tachycardia (heart rate > 100 beats/m in) were present the correspondent 35 days mortality rates were 54% and 61%. In regards to the choice of the rest thrombolytic agent in the setting of cardiogenic shock the international study[27] found a higher mortality rate in patients

treated with recombinant tissue plasminogen activator (r-TPA) compared with streptokinase (78% vs 68%). Similar data are reported by GUSTO-1 study (30 days mortality rate 57% for patients treated with r-TPA vs 51% for patients treated with streptokinase)[28].

In the society for cardiac angiography registry mortality rate was 72% in reperfused patients and 84% in those without reperfusion. In the Duke series, in hospital mortality rate was 30% for patients treated with thrombolysis of infarct related artery at the time of catheterization.

To improve the results of thrombolysis, an aggressive use of vasopressors or intra-aortic balloon pump (IABP) has been advocated. The consequent increase in blood pressure would have the potential to restore the rate of thrombus dissolution to normal values[29,30].

In the **GUSTO-1 study**[31], in which all patients underwent a thrombolytic regimen, early IABP institution was associated with a trend toward lower 30-days and 1-year mortality rates. In a retrospective evaluation of the National Registry of Myocardial Infarction the use of IABP in combination with thrombolytic therapy in patients with Acute Myocardial Infarction complicated by cardiogenic shock was associated with a marked reduction in mortality rate[32], conversely, a benefit was not seen in patients undergoing coronary angioplasty. In the SHOCK trial registry 27, thrombolysis and IABP were associated with a 17% and 32% lower mortality rate, respectively, and a 39% lower mortality rate when combined in comparison with the mortality rate observed in patients not treated with either thrombolysis or IABP.

RIGHT VENTRICULAR INFARCTION

For a clinician myocardial infarction meant infarction of the "Left Ventricle" only till 1974, when Cohn et at for the first time described potentially serious and unique haemodynamic consequences of right ventricular infarction. About 2/3 of patients to the hospital with Acute Myocardial Infarction show the ECG evidence of infarction of the anterior wall of left ventricle and approximately one third show the evidence of inferior wall Myocardial Infarction. Right ventricular infarction has been reported to occur in about 1/3 of the cases of inferior wall Myocardial Infarction, Isolated Right Ventricular Infarction also does occur and has been reported to be in less than 2% of cases of Acute Myocardial Infarction patients seen on autopsy.

Various pathological studies reveal that Right Ventricular Infarction is present in 14 to 34% of patients with transmural left ventricular infarction. The evidence of Right Ventricular Infarction as compared to left ventricular infarction is low, the possible mechanisms are –

1. The smaller mass of right ventricle
2. The lower tension within the right ventricular wall
3. The richer collateral circulation of right ventricle
4. The thickness of the right ventricular wall perhaps enables the chamber to derive relatively more nutrition from blood within its cavity.

normal descent of right ventricle base virtually excludes haemodynamically significant RVMI.

Mechanical Complications of Myocardial Infarction

Includes Ventricular septal defect, free wall rupture, papillary muscle rupture as a group mortality due to these complications are about 15% from Acute Myocardial Infarction.

Rupture of the free wall of the infarcted ventricle occurs in upto 10% of patients dying in the hospital of Acute Myocardial Infarction.

Factor responsible for rupture are :-

1. Thinness of the apical wall
2. Marked intensity of necrosis
3. Poor collateral flow
4. The shearing effect of muscular contraction against an inert and stiffened necrotic area.
5. Aging of the myocardium with laceration of the myocardial microstructure.

Have all been responsible for rupture.

It occurs more, frequently in the elderly & women and appears to be more common in hypertensive than in normotensive patients. It occurs approximately 7 times more frequently in the left than the right ventricle and seldom occurs in the atria. Usually involve the anterior or lateral wall of the ventricle in the area of

In approximately 30% of the patients (20%-45%) with inferior left ventricular wall myocardial infarction there is some evidence of R.V. necrosis.

The classic diagnostic feature of RVMI in ECG is 1 mm or more of ST elevation in the right precordial leads from V_3R to V_6R, however it is more specific in V_4 R. The ST segment, which is higher in lead V4R than in leads V_1 and V_3R offers the highest specificity and sensitivity in diagnosis. The overall specificity and sensitivity is 80% to 90%. Echocardiography helps to assess the site extent and severity of right ventricle wall motion abnormality. The sensitivity and specificity for diagnosis are about 82% and 93% respectively[33].

Goldberger[34] studied 24 cases of right ventricle Myocardial Infarction of which 9 were haemodynamically significant i.e. JVP>17 cm H_2O or a RA pressure of >13 mmHg. The descent of right ventricle base (a measure of right ventricle ejection fraction) was 0.7 + 2 cm in systole as compared to 1.3 + 0.4 cm in patients with non haemodynamically significant RVMI and 2+0.2 cm in normals. The sensitivity of this finding was 100% and specificity was 80%. The respiratory caval index (% collapse of inferior vena cava with inspiration) an index of increased RA pressure, was 22% $\pm$ 11% in haemodynamically significant RVMI as compared to 45% $\pm$ 15% in patients with haemodynamical stability. Finally an increased ratio of right ventricle to left ventricle end diastolic dimension greater than 0.9 was 50% sensitive & 87% specific for the haemodynamically compromised. The clinical implication of this study indicated that haemodynamically most useful of these measurements is the descent of right ventricle base. A normal respiratory caval index or a

distribution of left anterior descending coronary artery. Commonly associated with large transmural infarction involving at least 20% of the left ventricle. It can occur between 1st day & 3 weeks but most commonly 1 to 4 days following infarction and usually occurs near the junction of the infracted and normal muscle.

Rupture of the free wall of the left ventricle usually leads to hemopericardium and death from cardiac tamponade. Occasionally, rupture of the free wall of the ventricle occurs as the first clinical manifestation in patients with undetected or silent myocardial infarction and then it may be considered a form of "Sudden Cardiac death".

Incomplete rupture of the heart may occur when organizing thrombus and hematoma, together with pericardium, seal a rupture of the left ventricle and thus prevent the development of hemopericardium. With time this area of organised thrombus & pericardium become a pseudoaneurysm/false aneurysm that maintains communication with the cavity of the left ventricle in contrast to true aneurysm which always contain some myocardial elements in their walls. The walls of pseudoaneurysm are composed of organised hematoma and pericardium and lack any elements of the original myocardial wall. Pseudo aneurysm can become quite large even equaling the true ventricular cavity through a narrow neck. Frequently pseudoaneurysm contain significant quantities of old and recent thrombus which can cause arterial emboli.

Cabral S et al[35], (1998), reported that post Myocardial infarction left ventricular pseudoaneurysm resulting from free wall rupture is a very rare finding. Its recognition during life is even rarer.

Definitive preoperative diagnosis is difficult. A case of a left ventricular pseudoaneurysm as a mechanical complication of Acute Myocardial Infarction was diagnosed by transthoracic echocardiography.

Mesa Garcia JM et al36, 1998 reported that Mechanical complication after an Acute Myocardial Infarction, specially the subacute ventricular rupture was the most frequent complication. Mechanical complications constitute the second cause of death after Acute Myocardial Infarction following pump failure. The most frequent mechanical complication is ventricular rupture, which is the cause of death in 26% of the cases of Acute Myocardial Infarction. The incidence of septal & papillary muscle rupture is considerably less frequent.

Tate Da et al37, 1998 reported that ventricular free wall rupture is a well known catastrophy of Acute Myocardial Infarction. A significant number of patients present in a subacute fashion and can be successfully treated with surgery if diagnosed promptly. They presented a case of subacute free wall rupture that occurred after an undiagnosed myocardial infarction. The findings at pericardio centesis were unusual in that the fluid was sanguinous but not frank hemopericardium. This patient represents the first known reported case to present without frank hemopericardium who survived and was successfully treated surgically. The absence of frank hemopericardium showed not exclude the diagnosis of free wall rupture.

Tikiz H et al38, 2001 had done study on 350 consecutive patients suffering from first attack of Acute Myocardial Infarction.

The study aimed to determine the independent factors involved in the development of left ventricular aneurysm in Acute Myocardial Infarction, The overall incidence of left ventricular aneurysm was 11.7% (41/300) and no statistical difference was found between the incidence of left ventricular aneurysm between the 2 group i.e. thrombolytic group and control group. In univariate analysis, vessel patency, proximal left anterior descending artery (left anterior descending) stenosis, total left anterior descending occlusion, multivessel disease and hypertension were found to be important factors in left ventricular aneurysm formation after Acute Myocardial Infarction. Conclusion of the study is that not all patients who received thrombolytic therapy, but only those with PIRA (Patent infarct related artery) had evidently reduced incidence of left ventricular aneurysm. Patients with total occlusion with proximal left anterior descending stenosis and without PIRA (Patent infarct related artery) were found to have increased risk for formation of left ventricular aneurysm after Acute Myocardial Infarction. These findings indicate that the presence of vessel patency has a preventive effect on left ventricular aneurysm formation in Acute Myocardial Infarction.

The incidence of rupture of the interventricular septum is probably in the range of 2% of Acute Myocardial Infarction patients because death usually is not immediate and patients frequently can reach the hospital. Clinical features associated with an increased risk of rupture of the IVS (inter ventricular septum), include lack of development of a collateral network, advanced age, hypertension and possibly thrombolysis. The perforation may be a direct through and through opening, or it may be more irregular and serpiginous. The size of the defect determines the magnitude of the left to right

shunt and the extent of hemodynamic deterioration. Rupture of the septum with an anterior infarction tends to be apical in location, whereas inferior infarction are associated with perforation of the basal septum and with a worse than those in an interior location. Clinically it presents with a new harsh, loud holosystolic murmur that is best heard at the left lower sternal border and usually accompanied by thrill. Biventricular failure generally ensues within lows to days. The defect can also be recognized by 2-dimensional echocardiography with colour flow doppler imaging or insertion of a pulmonary artery balloon catheter to document the left to right shunt.

Di Sunama et al39 1997 had reviewed 34 patients complicating Acute Myocardial Infarction. They reported ventricular septal defect represents a serious complication after Acute Myocardial Infarction with an incidence 1-2%.

Cooley DA40, 1998 reported the post infarction ventricular septal rupture is an uncommon but serious complication of Acute Myocardial Infarction.

Mesa Garcia J.M. et al36 1998, reported that Mechanical complication after an acute Myocardial Infarction, specially the subacute ventricular rupture was the most frequent complication. Mechanical complication constitute the second cause of death after Acute Myocardial Infarction following pump failure. The most frequent mechanical complication is ventricular rupture which is the cause of death in 26% of the cases of Acute Myocardial Infarction. The incidence of septal & papillary muscle rupture is considerably less frequent.

Partial or total rupture of a papillary muscle is rare but often fatal complication of transmural Myocardial Infarction. Inferior wall infarction can lead to rupture of the posteromedial papillary muscle which occurs more commonly than rupture of the antero lateral muscle, a consequence of anterolateral Myocardial Infarction. Rupture of a right ventricular papillary muscle is rare but can cause massive tricuspid regurgitation and right ventricular failure. Complete transection of a left ventricular papillary muscle is incompatible with life because the sudden massive mitral regurgitation that develops can not be tolerated. Rupture of a portion of papillary muscle, usually the tip or head of muscle, resulting in severe mitral regurgitation, is much more frequent and is not immediately fatal. Unlike rupture of the ventricular septum, which occurs with large infarcts, papillary muscle rupture occurs with a relatively small infarction in approximately one of the case seen in the patients with papillary muscle rupture manifest a new holosystolic murmur and develop increasingly severe heart failure. In both conditions i.e. ventricular septum defect & mitral regurgitation the murmur may become softer or disappear as arterial pressure fall. Mitral regurgitation due to partial or complete rupture of a papillary muscle may be promptly recognized echocardiographically. Color flow Doppler imaging is particularly helpful in distinguishing acute mitral regurgitation from ventricular septal defect in the setting of Acute Myocardial Infarction.

Villavicencio R et al41 1991, reported that mitral regurgitation was due to valve ring dilatation with an increase of left ventricle diameter and decrease on ventricular systolic function.

Curcio Ruigomez et al42 1997 studied a case of double posterior Acute Myocardial Infarction complication and find ventricular septal defect, Ventricular aneurysm & acute and severe mitral regurgitation and the patients developed pulmonary hypertension.

Honma H et al43 1997, Tokyo Japan Evaluated 223 patients of Acute Myocardial Infarction by colour doppler echo. Mitral regurgitation present in 21% of the patients at the onset & developed in 18% of the patients during follow up. The group with unsuccessful recanalization and un-improved mitral regurgitation showed a significantly greater left ventricular end diastolic volume as well as lower left ventricular fraction than the patients with successful recanalization and no Mitral regurgitation. The results suggest that successful recanalization after myocardial infarction may decrease the incidence of mitral regurgitation and may prevent left ventricle remodeling resulting in a secondary improvement of mitral regurgitation.

Benico Barzilia et al in their study documented that only 43% of the total patient with mitral regurgitation detectable by doppler had murmur suggestive of mitral regurgitation when auscultated by an experienced observer, 17% of patients without mitral regurgitation had similar murmur. It may not be surprising that mitral regurgitation increase in incidence between 30 days and 3 months post infarction where remodeling is though to be more extensive. The lack of a relation to the extension of infarction makes ischemia/infarction of papillary muscle, which is thought to be more frequent in patients with inferior wall infarction.

Chuttane S.K. et al[44] studied 46 patients of Acute Myocardial Infarction with hemodynamic impairment 46% of the total group had echo proved mitral regurgitation while only 24% of them had regurgitation clinically. According to Killip classification 1st class had 25%, 2nd class 44.44%, 3rd and 4th class showed 100% of the clinical mitral regurgitation/echo mitral regurgitation. And as the time passes the tendency of mitral regurgitation increase as during 2nd visit (8th weeks after discharge) 43% of the patients of Killip class 1st and 2nd, and the patients of Killip class 3rd and 4th either died at admission or during follow up.

Yanagi H, et al[45], 1998 reported a case of acute mitral regurgitation caused by complete posterior papillary muscle rupture as a complication of acute inferior myocardial infarction. A 64 years old women developed sudden, cardiogenic shock shortly after the onset of acute inferior Myocardial infarction, with posterior papillary muscle totally ruptured.

Moustapha A, et al[46] 2001 present a case of an Acute Myocardial Infarction presenting solely as rupture of the head of antero-lateral papillary muscle of the mitral valve with on echocardiographic appearance of a mitral valve vegetation. A 61 Year old male Patients presented to the hospital with cardiogenic shock. Transesophageal echocardiography revealed function with the echo-cardiographic appearance of a large vegetation attached to the anterior mitral valve leaflet & severe mitral regurgitation.

ARRHYTHMIAS :

The incidence of Arrhythmias is higher in those patients seen earlier after the onset of symptom. Many serous Arrhythmias

develop before hospitalization even before the patient is monitored[47]. They include ventricular arrhythmias, Supraventricular arrhythmias, bradyarrhythmias.

Electrical instability : leads to ventricular premature beats ventricular tachycardia ventricular fibrillation, accelerated idioventricular rhythm & non paroxysmal A-V-junctional tachycardia.

Pump failure / Excessive sympathetic stimulation is responsible for sinus tachycardia atrial fibrillation, atrial flutter or paroxysmal supraventricular tachycardia.

Bradyarrhythmias and conduction disturbances include sinus bradycardia, junctional escape rhythm, atrio-ventricular block and intra ventricular block.

The conventional definition of ventricular ectopic impulses at rate of 120 or greater. They are of 2 type; *sustained* and *non-sustained* ventricular tachycardia. The sustained ventricular tachycardia lasting for 30 seconds of more or associated with hemodyanamic compromise, while non sustained tachycardia lasts for less than 30 seconds.

Prevalence of NSVT varies in relation to timing of Myocardial Infarction, within first 24 hours in pre thrombolytic ear, the prevalence of NSVT was 45% which is 75% now in thrombolysed patients[48]. The prevalence of NSVT during late hospital phase (10-30 days) in prethrombolytic-era was 7%-16% compared to 7-9% in thrombolysed patients.

Their incidence remain fairly constant over the first year of infarction. The most important factor associated with increased occurrence of NSVT is low LVEF and approximately 3/4 of patients with NSVT EF < 40%[48,49]. Other associated factors are multivessel disease, increased regional wall motion abnormality, inducible ischemia, ventricular aneurysm and abnormal hemodynamic[48,50]. NSVT detected 10-30 days after acute infarction more than double the risk of subsequent sudden death compared to patients without NSVT. NSVT detected 3 month to 1 years post Myocardial Infarction is also associated with a significant higher mortality rate.

MUSTT[51] **(Multicentre Unsustained Tachycardia Trial)** has shown that the risk of sudden death was reduced by 27% in patients randomized to electrophysiologically guided therapy. Patients randomized to electrophysiologically guided therapy who failed to responded to pharmacologic antiarrhythmic therapy, judged by serial electrophysiologic tests and thereby with implantable cardioverter defibrillators (ICD) experienced a 76% reduction in the risk of sudden death as compared to patients with no antiarrhythmic therapy. A recently published study **MADIT (Automatic Defibrillator Implantation Trials)**[52] has shown that post Myocardial Infarction patients with reduced left ventricular ejection fraction < 0.35 and non suppressible NSVT with procainamide treated with ICD have 54% reduction in mortality compared to amiodarone therapy. A 33% reduction in mortality was also obtained in patients who were on antiarrhythmic therapy and whose VT suppressed by these drugs. Thus does considering results of these trials patients with coronary artery disease, abnormal ejection and symptomatic NSVT are at high risk of sudden death which can be

reduced with EPS guided anti arrhythemic drug therapy and more effectively by ICD[48].

The sustained ventricular tachycardia may develop in specialized conduction system distal to bundle of His, in ventricular myocardium, or by an interaction between two. It occur at heart rate of > than 100 beats / min and last for 30 seconds or more associated with hemodynamic compromise[53,54,55]. Most common underlying heart disease is coronary artery disease accounting for more than 50% of cases followed by cardiomyopathy. Less, common causes include primary electrical disease, MVP, Valvular heart disease, congenital heart disease and miscellaneous causes[55,56]. Left ventricular hypertrophy and transient artery spasm may also lead to VT. Complex can occur after CABG sustained ventricular tachycardia are more likely to have reduced ejection fraction, slow conduction and ECG-abnormality left ventricle aneurysm, the abnormal signal average ECG, and previous Myocardial Infarction that patients with ventricular fibrillation[53,55,56].

Patients with sustained ventricular tachycardia or Ventricular fibrillation in the absence of reversible or transient cause has sudden death 30% and 2 years of sudden death of 50%[55,57].

Bobrov VA et al[58] studied 85 patients with acute large size myocardial infarction admitted within 12 hours of the condition development. 24 hours, monitoring was instituted. The result obtained showed only 27% of patients at low risk development of life threatening arrhythmias in all others high grades ventricular extrasystole are recorded including ventricular tachycardia in 50% of the patients.

Atrial fibrillation is the most common supraventricular arrhythmia in patients with Acute Myocardial Infarction.

Denes P et al[59] studied 1,211 patients with Acute Myocardial Infarction. Patients with bundle branch block were excluded from the analysis and remaining 1,158 were followed for upto 1 year after infarction out of them 45 patients had a serious arrhythmic event.

Eldar M et al[60]***, 1998***, had observed that paroxysmal atrial fibrillation is considered a frequent complication of Acute Myocardial Infarction associated with increased in hospital stay and long term mortality rates. The incidence of paroxysmal atrial fibrillation was (8.9% - 9.9%) and after 30 days (25.1%-27.6%) and at 1 year (38.4%-42.5%).

Pizzetti F et al[61]***, 2001***, data derived from GISSI – 3 trial, wherein included 17944 patients within the first 24 hrs. after Acute Myocardial Infarction. Atrial fibrillation was recorded during the hospital stay, and followup visits were planned at 6 weeks & 6 months. Survival of the patients at 4 years was assessed through census offices has done. The incidence of in hospital atrial fibrillation or flutter was 7.8%. Atrial fibrillation was associated with indicators of a worse prognoses (age > 70 years, female sex higher Killip class, previous myocardial infarction treated hypertension, high systolic BP at admission, IDDM, signs or symptoms of heart failure) and with some adverse clinical events (reinfarction, sustained ventricular tachycardia, ventricular fibrillation).

After adjustment for other prognostic factors, atrial fibrillation remained an independent predictor of A increased in hospital mortality 12.6% vs 5%. Long term mortality i.e. 4 years after Acute Myocardial Infarction confirm the persistent negative influence of AF. So conclusion is that atrial fibrillation is an indicator of worse prognosis after Acute Myocardial Infarction in the long term even in un selected population.

Ischemic injury can produce conduction block at any level of the AV or intraventricular node and the bundle of His, producing various grades of atrio -ventricular block.

Altun A, et al[62] 1998 reported after the study of 51 patients with inferior wall Acute Myocardial Infarction that advanced atrioventricular block is a frequent complication in patients in hospital, it occurs concurrently, with other complications and is associated with high mortality.

Kosuzue M, et al[63] 2001 reported early complete atrio ventricular block in patients with reperfused inferior wall Acute Myocardial Infarction.

Ozdemir K, et al[64] 2001, studied 172 patients (141 men & 31 women) between 28 & 84 years of age with acute inferior wall infarction out of 172,25 patients developed left ant hemiblock (LAHB). LAHB development during acute inferior-myocardial infarction can be indicator of left anterior descending lesion.

RECURRENT CHEST DISCOMFORT :

Post infarction recurrent chest discomfort may be due to recurrent chest discomfort, recurrent angina or infarction from non ischemic causes of discomfort that might be caused by infarct expansion, pericarditis, pulmonary embolism and non cardiac conditions. Important diagnostic maneuvers include a repeat physical examination, repeat ECG and assessment of the response to sublingual nitroglycerin, 0.4 mg.

The incidence of post infarction angina without reinfarction is between 20% and 30%[65]. It does not appear to be reduced by thrombolytic therapy[66,67] but has been reported to be lower in patients who undergo primary PTCA for Acute Myocardial Infarction. Usually extension and reinfarction refer collectively under the more general term, **recurrent infarction**[68]. Extension of the original zone of necrosis or reinfarction in a separate myocardial zone is difficult to differentiate within the first 24 hours after index event. Beyond the first 24 hours, when serum cardiac marker such as CK-MB have usually returned to the normal range, recurrent infarction may be diagnosed either by re-elevation of the CK-MB above the upper limit of normal and increased by at least 50% of the previous value or the appearance of new Q waves on the ECG. The incidence of this complication of Acute Myocardial Infarction range from about 5% to as a higher as 20% within the first 6 weeks and may be some what higher in patients who have received thrombolytic therapy. ***Marmor*** reported that recurrent infarction occurred frequently in obese females and was most common in patients with diabetes mellitus, those with a previous Myocardial Infarction and those with an early peaking CK-MB curve (< 15 hours) but it is not predictable from the

angiographic appearance of the coronary artery early after infarction.

Regardless of whether post infarction angina is persistent or limited, its presence is important because short-term morbidity is higher among such patients, mortality may be increased if the recurrent ischemia is accompanied by ECG changes and hemodynamic compromise recurrent infarction (due in many cases to reocclusion of the infarct related coronary artery) caries serious adverse prognostic information because it is associated with a two to four fold higher rate of in hospital complication (CHF, heart block) and mortality. The mortality rate at 1 to 3 years following the initial infarction is higher in those patients who suffered from recurrent infarction during their index hospitalization. Presumably the higher mortality is related to the larger mass of myocardium whose function become compromised.

PERICARDIAL EFFUSION AND PERICARDITIS :

Pericardial effusion occur in approximately 25% of patients after Myocardial Infarction effusion are more common in patients with anterior wall Myocardial Infarction and with large infarcts and when congestive failure is present. The majority of pericardial effusion that are seen following Acute Myocardial Infarction do not cause hemodynamic compromise. Cardiac tamponade occur usually due to ventricular rupture or hemorrhagic pericarditis.

The reabsorption rate of a post infarction pericardial effusion is slow, with resolution often taking several months. The presence of an effusion does not indicate that pericarditis is present, although

they may occur together, the majority of effusion occur without other evidence of pericarditis.

Pericarditis, when secondary to transmural Acute Myocardial Infarction, may produce pain as early as the first day and as late as 6 weeks after Myocardial Infarction. The pain of pericarditis may be confused with post infarction angina, recurrent infarction or both. An important distinguishing feature is the radiation of the pain to either trapezius ridge, a finding that is nearly pathognomonic of pericarditis and rarely seen with ischemic discomfort. Transmural Myocardial Infarction by definition, extends to the epicardial surface and is responsible for local pericardial inflammation. An acute fibrinous pericarditis **(Pericarditis episteno cardica)** occurs most commonly after transmural infarction. Although transient pericardial friction rubs are relatively common among patients with transmural infarction within the first 48 hrs. pericardial friction rub appears to be correlated with a larger infarct and greater hemodynamic compromise. The discomfort of pericarditis usually becomes worse during a deep inspiration, but it may be relieved or diminished when the patients sits up and leans forward. Although anticoagulation clearly increases the risk for hemorrhagic pericarditis early after Myocardial Infarction, this complication has not been reported with sufficient frequency, but the detection of a pericardial effusion on echo is usually an indication for discontinuation of anti-co-agulation.

Dressler Syndrome also known as the post myocardial infarction syndrome, usually occurs 1 to 8 weeks after infarction. Dressler cited an incidence of 3 to 4% of all Acute Myocardial Infarction patients in 1957 but the incidence has decreased dramatically since that time. Clinical features of syndrome are

malaise, fever, pericardial discomfort, leukocytosis, an elevated sedimentation rate and a pericardial localized fibrinous pericarditis containing polymorphonuclear leukocytes. The detection of antibodies to cardiac tissue has raised the notion of an immunopathological process.

Cerebrovascular accidents : clinically apparent thromboembolism complicates Acute Myocardial Infarction in 10% of cases, but in 20% of patients it is clinically silent. Thromboembolism is considered to be least an important contributing cause of deaths in 25% of patients with Acute Myocardial Infarction who die after admission to the hospital. Arterial emboli originate from left ventricle mural thrombi, while most pulmonary emboli arise in the leg veins.

Thromboembolism typically occurs in association with large infarct (especially anterior) CHF and a left ventricle thrombus detected by echocardiography 2-D echocardiography reveals left ventricle thrombi in about 1/3 of patients with anterior wall infarction but in few patients with inferior wall infarction. Arterial embolism often presents as a major complication, such as hemiparesis when the cerebral circulation is involved in hypertension if the renal circulation is compromised. The incidence of embolic complication appears to be markedly lowered by such therapy.

Intracranial hemorrhage is an uncommon but very dangerous complication in patients receiving thrombolytic therapy for Acute Myocardial Infarction. Neuro surgical evacuation is often an available treatment option. GUSTO-1 trial randomly assigned 41,021 patients with Acute Myocardial Infarction to 1 to 4 thrombolytic strategic in

1081 hospitals in 15 countries. A total of 268 patients (0.65%) had an intra cranial hemorrhage. ***Mahaffey KW et al***[69] assessed difference in clinical characteristic, neuro imaging features, glasgow coma scale scores, functional status (disable : moderate or severe deficit, not disable; no or minor deficit) a 30 days mortality rate between the 46 patients who underwent neuro surgical evacuation and the 222 patients who did not. Mortality rate at 30 days for all patients with intracranial hemorrhage was 60%, an additional 27% were disabled. Evacuation was associated with significantly higher 30-day survival (65% versus 35%) and a improved functional status (non disabling stroke : 20% versus 12%) conclusion is that although intracranial hemorrhage is uncommon after thrombolysis for Acute Myocardial Infarction, 87% of patients die or have disabling stroke.

ELECTRO CARDIO GRAM

It is electrical device which records the changing potentials of the electrical field imparted by heart. It is extremely useful clinical laboratory tool and it the only practical means of recording the electrical behaviour of the heart so ECG serves as a gold standard for the diagnosis of arrhythmias.

The electro cardiographic recording paper is divided into small and large squares. The small squares are 1mm square. The large squares are 5mm squares. The square facilitate the measurement of (I) time parameters (horizontal measurement) and (ii) Deflexion amplitude (vertical measurement). Conventionally electrocardiogram is nearly always recorded at a paper speed of 25mm per second. At this paper speed 5 large squares represent one second and one small square represents 0.04 second. Most graph papers used for the recording of ECG have every 15th large squarel 3 sec. Period l marked by a vertical line on the upper border.

The conventional electrocardiographic leads :

In clinical practice, however there are 12 conventional leads, divided into 2 groups.

1. ***The Frontal plane leads*** – These are oriented in frontal or coronal plane of the body I, II, III, aVR, aVL and aVF
2. ***The horizontal plane leads*** – These are oriented in the transverse or horizontal plane of the body and are formed by the precordial leads V_1 to V_6 .

The frontal plane leads :

Standard lead I – This lead is derived from the placement of negative electrode on the right arm and the positive electrode on the left arm.

Standard lead II – This lead is derived from the placement of the negative electrode on the right arm and the positive electrode on the left foot.

Standard lead III – This lead is derived from the placement of the negative electrode on the left arm and the positive electrode on the left foot.

The lead axes of these 3 leads form a triangle and as the electrodes of these leads are regarded as equidistant from the heart, the lead axes too may be considered to be equidistant from the heart. These lead axes thus form an equilateral triangle with the heart at the centre – Einthoven's triangle.

The horizontal plane leads :

Lead V_1 - Placed over the 4th intercostal space immediately to the right of sternum.

Lead V^2 - Placed over the 4th intercostal space immediately to the left of sternum.

Lead V^4 - is placed over the 5th intercostal space in the mid clavicular line.

Lead V^3 - is placed on the chest exactly midway between the lead V_2 and lead V_4 electrode position.

Lead V^5 - is placed at the same horizontal level as lead V_4 but on the anterior axillary line.

Lead V^6 - is placed at the same horizontal level as lead V_4 and V_5 but on the mid axillary line.

Orientation of the conventional electrocardiographic leads :

- Standard lead II, III and AVF – oriented to the inferior surface of the heart.
- Standard lead I, AVL tend to be oriented to the high or superior left lateral wall.
- Lead aVR is oriented to the cavity of the heart.
- Lead V_1 also tends to be oriented towards the cavity of the heart.
- Leads V_1 to V_6 are oriented towards the anterior wall of the heart. These may be arbitrarily sub divided into :

  (i) Antero – septal leads – V_1 to V_4

  (ii) Apical or lateral leads – V_5 to V_6

  (iii) Lateral leads – I & AVL

- Leads V_1 & V_2 tend to be oriented to the right ventricle
- Leads V_4 & V_6 tends to be oriented to the left ventricle
- There is no lead which is oriented directly to the posterior wall of the heart.

Genesis of the normal ECG :

P wave = due to atrial depolarization

QRS complex = due to ventricular depolarization
T wave and 'U' wave = due to ventricular depolarization 'U' wave best seen in leads V_2 to V_4

ECHOCARDIOGRAPHY

The term echocardiography refer to a group of tests that utilize ultrasound to examine the heart and record information in the form of echoes i.e. reflected sonic waves. The sonic frequency used for echocardiography ranges from 1 to 10 million cycles/second or 1 to 10 mega Hertz (MHz) while in children they are usually higher ranging from 3 to 10 MHz. Whereas a barrel chested, emphysematus patients needs a 1.6 MHz transducer. The resolution of the recording which is the ability to distinguish 2 objects that are spatially close together, varies directly with the frequency and inversely with the wavelength. Waves passes radially through liquid, such as blood or pericardial fluid and these are displayed as **black** on 2 dimensional image. When ultrasound is reflected off more solid structures, such as the myocardium and valves, these is **gray scale** display. Structure such as calcium produce intense acoustic reflection and are displayed as bright **white** on 2D-image.

M-mode : Previously M-mode (time motion-mode) echo cardiography was used. M-mode echocardiography provides information about –

- fractional shortening

- ejection fraction
- septal and wall thickness and
- left ventricle mass

The M-mode technique is limited that it provides only a one dimensional view of the heart.

2D Echocardiography : It has allowed cardiac structure to be visualised in a real time fashion with the help of this we can now assess intra cardiac lessons observe (I) contractility & (II) valvular function.

Doppler echocardiography : It utilizes ultrasound to record blood flow within the cardiovascular system.

Echocardiography can be performed in 2 ways e.g.

(i) Transthoracic **(ii) Transesophageal**

Transthoracic : Echocardiogram, the imaging is performed with a hand held transducer placed directly on the chest wall.

Transesophageal : Echocardiogram may be performed in which an ultrasound transducer is mounted on the tip of an endoscope placed in the esophagus and directed towards the cardiac structure, so that high resolution images of the posterior structures are obtained.

Method

The imaging is performed from multiple acoustic windows with different transducer location so that the entire heart and great

vessels can be displayed in real time and in various 2 dimensional planes. The patients are generally examined in the supine or left lateral semidecubitus position. Cardiac window is usually found between 3rd to 5th intercostal space slightly to the left of the sternal border.

Views : Standard cross sectional imaging planes are as following

1. Parasternal

(a) Long axis- Left heart : aortic valve mitral valve & left ventricle.
- Right ventricular inflow tract
- Right ventricular out flow tract
- Main pulmonary actery
- Cardiac apex

(b) Short axis- Aortic valve & left atrium
- Left ventricle (Mitral valve level)
- Left ventricle (papillary muscle level)
- Left ventricle apex

2. Apical
- *4 chamber*
- *5 chamber*
- *2 chamber*
- *long axis (left ventricle)*

3. Sub costal- *Long axis of the heart*
- *Long axis of the right ventricular out flow tract.*

4. Suprasternal- *Long axis of the aortic arch*
- *Short axis of the aortic arch*

Indication of Echo : In coronary artery diseases are

(1) Diagnosis of coronary artery disease
(2) Estimation of infarct size
(3) In reperfusion therapy
(4) In detection of complications
(5) Management of hemodynamically compromise patients
(6) Determination of prognosis and post myocardial risk stratification
(7) Determination of myocardial viability.

Advantages of echo :

(1) It is non invasive & cost effective

(2) No radiation involved

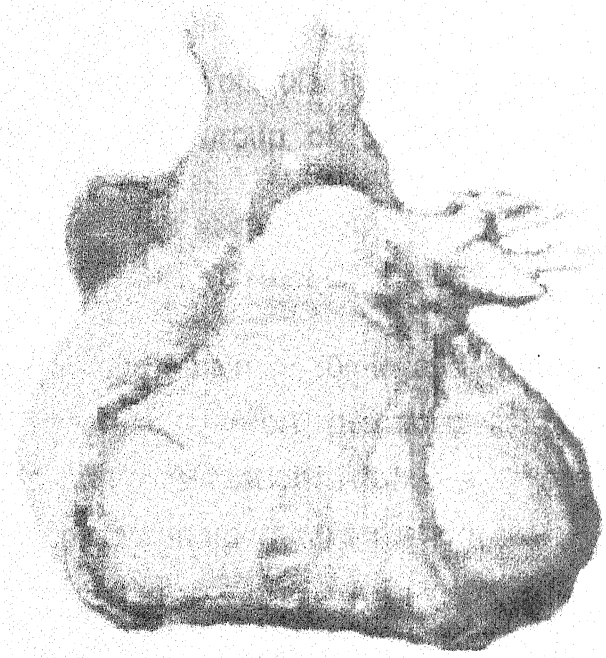
(3) Easy management

(4) Early diagnosis of myocardial infarction can be made even before enzyme level rises

(5) Associated diseases can be detected

(6) Detection of complications

(7) Prognosis & treatment can be selected.



MATERIAL AND METHODS

MATERIALS & METHODS

This study was conducted on the patients attending the cardiology clinic, emergency/out-patients department, ICCU & wards run by the Department of Medicine, M.L.B Medical College, Jhansi. The cases of Acute Myocardial Infarction were selected. The study was performed on group of 53 patients of Acute Myocardial Infarction.

CRITERIA'S OF SELECTION OF CASES :

1. **History** of chest pain > 30min and frequently for number of hours. Chest discomfort described as constricting, crushing, compressing sensation of a heavy weight or squeezing/stabbing/burning sensation.

2. **ECG finding –**

(a) ***Abnormal Q wave** > .03sec in lead I,II,III, aVI, aVF, V_4, V_5 V_6,
(Instead of amplitude duration of Q wave is important)

*Presence of Q wave in aVR, V_1, V_2, V_3, of any amplitude or duration.

(b) Abnormal 'R' wave

I	=	'R' amp $\leq$ 0.20mV
avl	=	'R' amp $\leq$ Q amp
aVF	=	'R' amp $\leq$ 2 x Q amp
V_1	=	'R' duration $\geq$ 0.04 sec
		'R' amp $\geq$ 0.60mv (6 small square)

'R' amp $\geq$ 'S' amp

V_2 = 'R' duration $\geq$ 0.05 sec
'R' amp $\geq$ 1.50mv (15 small sq) ▶ Post wall MI of LV
'R' amp $\geq$ 1.5 X 'S' amp

V_2 = 'R' duration $\leq$ 0.01 sec or amp $\leq$ 0.10mv

V_3 = 'R' duration $\leq$ 0.02 sec or amp $\leq$ 0.20mv

V_4 = 'R' duration $\leq$ 0.70 mv or $\leq$ 'Q' amp

V_5 = 'R' duration $\leq$ 0.70 mv or $\leq$ 2 x 'Q' amp

V_6 = 'R' duration $\leq$ 0.60 mv or $\leq$ 3 x 'Q' amp

(c) ST Segment –

- Slope elevation of the S - T segment
- ST segment become straightened with an upward slope

(d) 'T' wave –

'T' wave become very tall and wide

3. Cardiac Marker :

- Creatine phosphokinase – MB > 12iu/ltr. (95% specificity)
- Trop T +ve

4. Echo finding :

- RWMA - Regional Wall Motion Abnormality
- Decreased ejection fraction
- Fractional shortening
- Abnormal relaxation

History: The history was recorded in detail for each patient including duration of symptoms, personal history, past history, family history, dietary history, drug history & complications.

Examination : General examination was done to know the general condition, pulse rate, respiratory rate, blood pressure, temperature, pallor, icterus, cyanosis, clubbing, edema, hydration, lymphadenopathy, JVP etc. Multiple readings of blood pressure were taken at each visit, B.P. measured by mercury sphygnomanometer in both sitting & lying down position.

Systemic examination to find out changes due to myocardial infarction and associated complications, this included examination of neck, to palpate, auscultate the carotid and thyroid, examination of heart for size, rhythm & sound eg. associated murmur S_3 or S_4, pericardial rub etc.

Lungs for rhonchi and rales were examined. The abdomen was examined for hepatomegaly/hepatojugular reflux, renal masses, bruits over aorta or renal arteries, examination of extremities of peripheral pulses & Edema.

Neurological assessment was also done to detect hemiplegia or other neurological deficit.

Fundus examination was done to do evaluation of hypertensive or diabetic changes in eyes.

Investigations : The investigations included

1. Haemogram (Hb%, TLC, DLC, ESR)
2. Blood urea
3. Serum creatinine
4. Blood sugar (Fasting, P.P.)
5. Urine Routine
   Microscopic

6. Serial 12 leads – ECG

a. Qs complex
b. Qr complex
c. Loss of R wave amplitude
d. ST segment elevation
e. Tall & widened 'T' waves

7. CPK – MB
8. Trop – T
9. Lipid profile – S. Total cholesterol > 200mg/dl
S. Triglycerides
LDL > 160 mg/dl
HDL < 35

10. Serial Echocardiography

The patients were examined in left lateral position (To obtain a good echogenic window) 2-D and Doppler echocardiography examination were performed with HP- Sonos-2000 cardiac Ultrasound Unit using 2.5 MHz transducer. Measurement of different cardiac chambers were made according to the recommendations of the American Society of Echo-cardiography. The systolic and diastolic functions were examined as follows :

- Hypokinesia of wall (RWMA)
- Decreased ejection fraction < 50%
- Fractional shortening
- Abnormal relaxation pattern

a) E < A velocity
b) DT > 240 misc.

c) IRT > 100m.sec.

d) E/A ratio < 1

11. USG abdomen (if indicated)

12. X-Ray chest PA view in deep inspiration.

The cases were followed up during their stay in the hospital and subsequent to their discharge for variable periods of time during their routine visit to the OPD.

The data obtained were critically analysed & statistically evaluated.

WORKING PROFORMA

CLINICO-ECHOCADIOGRAPHIC EVALUATION OF PATIENTS OF ACUTE MYOCARDIAL INFARCTION

Investigator : **Dr. DEEPSHIKHA** (Medicine)
Maharani Laxmi Bai Medical College, Jhansi

- MRD/OPD No. :
- Place of investigation :
- Date of commencement of study :

1. Personal History

	Age/Sex	Religion	Education
1. Name			
2. Address			
3. Occupation			
4. Socio-economic status			
5. Rural/Urban			
6. Marital Status			

Daughter Son

2. H/O Coronary risk factors

	Duration	Quantity
1. Smoking		
2. Alcohol		
3. Diabetes		
4. Hypertension		
5. Elevated serum cholesterol		
6. Sedeutary habit		
7. Stress		

3. Complaints & present history

4. **Past history**
5. **Family history**
6. **Treatment history**
7. **Menstrual history (if female)**
8. **General examination**
   1. General appearance
   2. Weight
   3. Height
   4. Pulse rate, rhythm, volume character, radio temoral delay
   5. Blood pressure : sitting lying (systolic/diastolic in upper limbs)
   6. Pallor
   7. Icterus
   8. Cyanosis
   9. Clubbing
   10. Oedema
   11. Hydration
   12. HVP
   13. Skin-Xanthelasma/Tendon Xanthemia
   14. Eye : Arcus Seniles

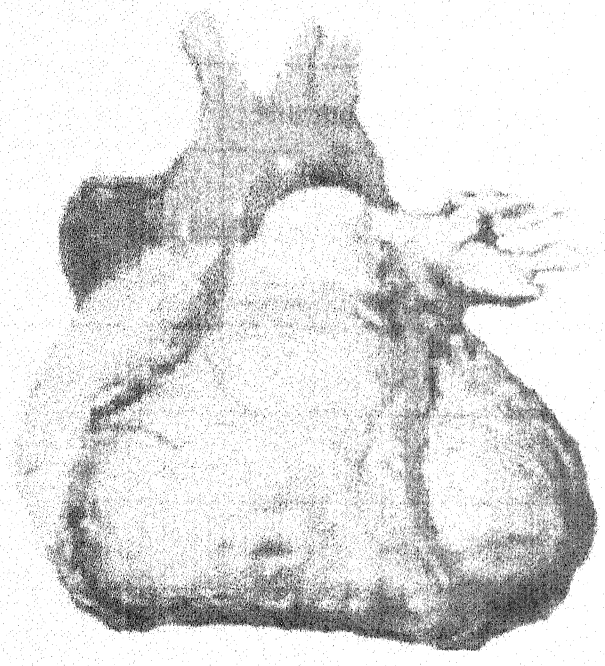
9. **Systemic Examination**
   (a) Cardiovascular system
   (b) Respiratory system
   (c) Abdomen
   (d) Central nervous system
   (e) Musculoskeletal system

10. **Investigation**
   - Hb%, TLC, DLC, ESR
   - B. sugar (F & PP)
   - B. urea
   - S. creatine
   - S.G.O.T
   - Lipid profile [s. cholesterol, HDL, LDL, S. triglyceride]
   - CPK – MB
   - Trop –T
   - ECG – 12leads
   - X – Ray chest PA view (in deep inspiration)
   - Echo
   - Fundus examination

11. **Advice**

12. **Follow-up**

Date	HR	PR	RR	BP	S_3, S_4	ST-T changes	CPK-MB	Complications	Others



OBSERVATION

OBSERVATION

TABLE - 1

Distribution of study subjects according to age (N=53)

Age in years	Number of patients	% of Total
<30	-	-
30-39	2	3.77
40-49	12	22.64
50-59	17	32.08
60-69	15	28.30
70-79	6	11.32
>80 yrs.	1	1.89
Total	53	100

Table – 1 shows that there were 53 patients in this group. Max patients were in 50-59 years age group.

TABLE - 2

Showing base line characteristics of all patients (N=53)

Characteristics	Male Patients	Female Patients	Total Patients
Number of patients	41	12	53
Age (Mean ± SD)	Mean 54.25 SD ± 10.56	59 SD ± 13.12	55.32 SD ± 11.22
Systolic B.P.			
(A) >160 MM Hz	15	1	16
(B) 140-159 mmHg	2	1	3
Diastolic B.P.			
(a) >100-109	9	-	9
(b) 95-99	6	2	8
Cigarette/Bidi Smoking	16	-	16
Tobacco Chewer	5	1	6
Alcoholic	6	-	6
Emotional Stress	6	1	7

H/O Oral Contraceptive	-	3	3
Family H/O Hypertension	3	3	6
Obesity	1	2	3
Family H/O Diabetes	3	1	4

TABLE NO - 3

Age & Sex distribution of Acute Myocardial Infarction Patients (N=53)

Age Group (Years)	Male		Female		Total	
	Number	%	Number	%	Number	%
<30	-		-		-	
30-39	1	1.88	1	1.88	2	3.77
40-49	11	20.75	1	1.88	12	22.64
50-59	15	28.30	2	3.77	17	32.07
60-69	11	20.75	4	7.54	15	28.30
70-79	3	5.66	3	5.66	6	11.32
>80 yrs.	-	-	1	1.88	1	1.88
Total	41	77.34 %	12	22.61 %	53	99.98

Table –3 Shows that there were total 53 patients in this group out of which 41 were males & 12 were females. Maximum number of patients was in age group 50-59 yrs. (32.07%). Male patients were maximum in age group 50-59 yrs. (28.30%) whereas female patients were maximum in age group 60-69 yrs. (7.54%).

TABLE – 4

Percentage of risk factors in male/female patients of Acute Myocardial Infarction (N=53)

Risk Factors	Male		Female		Total	
	No.	%	No.	%	No.	%
(1) Increased serum Cholesterol >200 mg/dl .	38	71.69	9	16.98	47	88.68
(2) Hypertension	17	32.07	2	3.77	19	35.84
(3) Smoking	16	30.18	-		16	30.18
(4) Diabetes Mellitus	7	13.20	2	3.77	9	16.98
(5) Stress	6	11.32	1	1.88	7	13.2
(6) Alcohol	6	11.32	-	-	6	11.32
(7) Tobacco chewer	5	9.43	1	1.88	6	11.32
(8) Sedentary habits	3	5.66	1	1.88	4	7.54
(9) Obesity	1	1.88	2	3.77	3	5.65
(10) Family history of Diabetes/Hypert.	1	1.88	1	1.88	2	3.76
(11) No risk factors	6	11.32	4	7.54	10	18.86

Table – 4 shows that there are 10 risk factor for Acute Myocardial Infarction patients. *Increase S. Cholesterol level* is present

in maximum number of patients in the present study which is 47 (88.68%) out of which 38 (71.69%) were male patients and 9 (16.98) were female patients. *Hypertension* is the second most common risk factor, which was present in 19 patients (35.84%), this group includes 17 male (32.07%) and 2 female (3.77%) patients. *Smoking* is the third important factor, present in 16 patients (30.18%) all were male. *Diabetes – Mellitus* as a risk factor present in 9 (16.98%) patients. Incidence of diabetes was more in males as compared to females, 7 & 2 respectively. *Alcohol* as a risk factor present only in males (11.32%) no female patient was found alcoholic. *Tobacco* as a coronary risk factors present in good number of patients 11.32% (6.53), it was also showing male predominance. *Stress* is also a significant risk factor for coronary artery disease in our study group, which is present in 13.2% (7/53) of patients.

TABLE – 5

shows Clinical presentation (%) in Acute Myocardial Infarction

(N = 53)

Clinical Presentation	Total Number	%
1. Chest Pain	42	79.25
2. Nausea & Vomiting	9	16.98
3. Dizziness	7	13.21
4. Weakness	9	16.98
5. Palpitation	27	50.94
6. Cold Perspiration	30	56.60
7. Sense of impending doom	38	71.69
8. Cerebrovascular Accidents (Cardio Embolic Stroke)	2	3.77

Table - 5 Symptoms which were commonly reported by Acute Myocardial Infarction patients are shown in Table-5. Chest pain was present in 42 patients (79.25). 38 patients (71.69%) were reported palpitation along with chest pain. Cold perspiration was present in 30 patients (56.60%). 27 patients (50.94%) gave the history of palpitation. Nausea & vomiting reported as common presenting symptoms in 9 patients (16.98%). Dizziness reported by 7 patients (13.21%).

TABLE - 6

Distribution of subjects according to site of infarction (N = 53)

ECG Changes	Total No.	%
1. Antero-Septal MI (V_1-V_4)	8	15.09
2. Antero lateral MI – (I, aVL, V_4, V_5, V_6)	18	33.96
3. Extensive Ant wall MI (I, aVL, V_1 to V_6)	9	16.98
4. Inf Wall MI (II, III aVF)	3	5.66
5. Infero post MI ($\pm$ RVI)	3	5.66
6. Ext Ant + Inf MI	9	16.98
7. Apical MI (V_5 & V_6)	-	-
8. RVH + RAD	1	1.89
9. WNL	2	3.77

Table 6 shows the electrocardiographic changes recorded in 53 patients. Antero – lateral Myocardial Infarction was present in 18 patients while 9 were presented with extensive Anterior wall Myocardial Infarction. 9 patients had extensive anterior & inferior wall Myocardial Infarction. 8 ECG showed changes of antero-septal Myocardial Infarction. Inferior wall Myocardial Infarction was Present in 3 patients. Infero post wall Myocardial Infarction reported in 3 patients out of which one had right ventricle infarction changes. 3 patients had showed no change in ECG but they were Trop T positive.

TABLE NO - 7

Showing Serum Total Cholesterol level (mg/dl) in study group (N=53)

Serum Total Cholesterol level (mg/dl)	Male	Female	Total	Percentage
150-169	2	2	4	7.55
170-199	1	1	2	3.77
200-219	9	1	10	18.87
220-240	9	3	12	22.64
>240	20	5	25	47.17
Total	41	12	53	100

Table 7 : Shows that the distribution of serum total cholesterol in various age groups. 47 patients (88.68%) of our study group had value >200 mg/dl out of which 25 patients (47.17%) were having value >240 mg/dl. The range of total cholesterol seen was 156-266mg/dl, and mean total cholesterol in our study was 208.88 mg/dl.

TABLE – 8

Shows Incidence of complications of Acute Myocardial Infarction

(N=53)

Complications	30-39 Yrs	40-49 Yrs	50-59 Yrs	60-69 Yrs	70-79 Yrs	>80 Yrs	Total	%
1.Ventricular dysfunction	2	9	12	8	4	-	35	66
2. Cardiogenic Shock	-	4	2	4	1	-	11	20
3. R.V. Infarction	-	1	-	1	-	-	2	3.77
4. VSD	-	-	1	-	-	-	1	1.88
5. MR	-	-	1	2	-	1	4	7.54
6. L.V. Aneurysm	-	1	-	-	-	-	1	1.88
7. Arrhythmias	1	-	2	2	2	-	7	13.2
8. A-V Block	-	1	2	1	1	-	5	9.4
9. Pericardial Effusion	-	3	3	2	1	1	10	18.86
10. Pericarditis	-	-	-	1	-	-	1	1.88
11. Thrombo Embolic episodes	-	-	1	-	1	-	2	3.77

12. Recurrent Chest pain	-	-	-	1	1	-	2	3.7
13. No Complication	-	1	1	2	-	-	4	7.54

Table 8 shows the different complications observed in the study group (N-53). The commonest was ventricular dysfunction with a total of 35 subjects out of 53 patients. 11 patients had cardiogenic shock. Pericardial effusion was present in 10 patients of Acute Myocardial Infarction. 7 subjects had arrhythmias & 5 developed A-V block following Myocardial Infarction. 4 patients had mitral regurgitation due to rupture of papillary muscle. Right ventricular infarction was detected in 2 subjects. Thromboembolic episodes & recurrent chest pain were present in 2 patients each while ventricular septum defect, left ventricle aneurysm, Pericarditis each was found in one patient.

TABLE - 9

Distribution of patients according to systolic & diastolic function following Acute Myocardial Infarction (N=53)

Age in Years	Systolic function with Normal diastolic function		Systolic dysfunction with normal diastolic function		Diastolic dysfunction with normal systolic function		Systolic + Diastolic dysfunction	
	No.	%	No.	%	No.	%	No.	%
>30	-	-	-	-	-	-	-	-
30-39	-		-		1	1.89	1	1.89
40-49	3	5.66	-		4	7.55	5	9.44
50-59	6	11.32	2	3.77	2	3.77	8	15.09
60-69	6	11.32	-		3	5.66	5	9.44
70-79	2	3.77	-		2	3.77	2	3.77
80-89	1	1.89	-		-		-	
Total	18	33.96	2	3.77	12	22.64	21	39.63

Table – 9 shows percentage distribution of systolic, diastolic and systolic + diastolic dysfunction in 53 patients of Acute Myocardial Infarction. 21 (39.63%) patients had systolic + diastolic dysfunction which is the largest group. Whereas 12 (22.64%) patients had isolated diastolic dysfunction. Isolated systolic dysfunction present only 2 patients while 18 patients showed normal systolic & diastolic dysfunction.

TABLE - 10

Distribution of arrhythmias according to their site of origin

(N = 53)

Age Groups	Arrhythmias					
	Atrial		Ventricular		Total	
	No.	%	No.	%	No.	%
<30 Yrs.	-	-	-	-	-	-
30-39 Yrs.	-	-	1	1.89	1	1.89
40-49 Yrs.	-	-	-	-	-	-
50-59 Yrs.	1	1.89	1	1.89	2	3.77
60-69 Yrs.	2	3.77	-	-	2	3.77
70-79 Yrs.	2	3.77	-	-	2	3.77
>80 Yrs.	-	-	-	-	-	-
Total	5	9.43	2	3.77	7	13.21

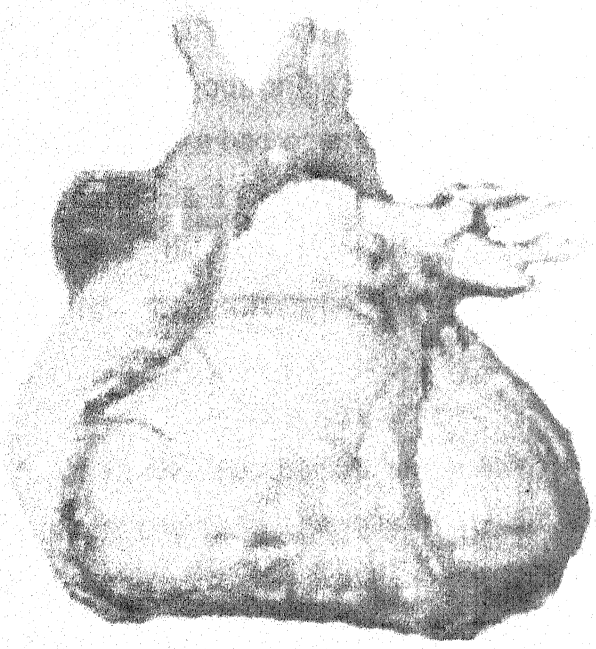
Table 10 shows, that out of 53 patients of Acute Myocardial Infarction, 7 patients developed arrhythmia. Ventricular arrhythmias presents in 2 patients (3.78%) while 5 patients reported as having atrial arrhythmia (9.43%).

7TABLE - 11

Distribution According to case mortality in Acute Myocardial Infarction from complications (N=53)

Complication	Number of case	Mortality	Case % (Mortality) rate
1. Ventricular dysfunction (Systolic/Diastolic)	35	6	17.14
2. Cardiogenic Shock	11	2	18.18
3. R.V. Infarction	2	1	50
4. VSD	1	-	-
5. MR	4	-	-
6. L.V. Aneurysm	1	-	-
7. Arrhythmias	7	2	28.57
8. A.V. block	5	-	-
9. Pericardial effusion	10	2	20
10. Pericarditis	1	-	-
11. Thromboembolic Episodes	2	2	100
12. Recurrent chest pain	2	-	-

Table –11 shows that mortality due to Acute Myocardial Infarction in 53 patients. 14 patients died due to post myocardial infarction. Out of 14 patients 6 were having severe ventricular dysfunction, 2 were having cardiogenic shock and 1 patient was died due to Right ventricular Infarction. Arrhythmias were responsible for death of 9 patients, Pericardial effusion was responsible for death of 2 patients. One patient died because of cardio embolic accident.



DISCUSSION

DISCUSSION

The present study has been carried out in cardiology unit of medicine department. Patients were included in this present study who were attending OPD, emergency, cardiology OPD & WARDS. The study has been carried out on 53 freshly diagnosed Acute Myocardial Infarction patients. Care had been taken by method of detail history, clinical examination & laboratory investigations to exclude those patients from the study who showed confounding factors for Acute Myocardial Infarction. Informed consent was taken from each patient.

Table –1 shows distribution of patients according to age. As can be seen from the table, the majority of patients were more than 50 yrs. of age which include 32.08% (17 out of 53). I have found no patients below the 30 yrs. of age. In age group 40-49 yrs. and 60-69 yrs. there are 12 & 15 patients respectively.

A study by ***Mammi MV, Parvitharan Rehman A et al[70], 1990*** at Calicut Medical college found that percentage of acute Myocardial Infarction patients below 40 yrs. were 17%. This figures has been 3.77% (2/53) in our study possibly because of small number of patients in our study. In this study they found out that 55% of the male patients of Acute Myocardial Infarction were below 50 yrs. This figure has been 22.64% in our study possibly because of the above reason. Percentage of patients below 55 yrs. in their study was 67% compared to 56.603% in our study.

In our study out of 53 patients of Acute Myocardial Infarction female patients were 12 i.e. 22.64%. This is a relatively low figure, the cause of which probably in small sample size of our patient population and also lower incidence and reporting of Myocardial Infarction in females of Bundelkhand region. The maximum number of female patients was in age group 60-69 that is in post menopausal age. The menopause was the probable cause of this.

In the present study out of 53 patients 41 were male and 12 were female. And mean age of patients were 55.32 SD $\pm$ 11.22. Mean age of the male patients were 54.25 SD $\pm$ 10.56 and female were having mean age was 59 SD $\pm$ 13.12.

In our study Hypertension as a risk factor was present in 35.84% (19/53) which was contributed by 32.07% male (17/53) & 3.77% female (2/53). Out of 19 patients; 17 patients were having both systolic and diastolic level more than normotensive range while two patients were having only systolic hypertension along with normal diastolic reading.

A study by J. Alasdair, Anthony F. Lever on pulse pressure as a predictor of cardiac risk in patients with Hypertension found that those with the greatest pulse pressure were at higher risk of coronary events across the range of systolic BP but the effect was most notable when systolic B.P. was high. In our study 19 patients were having high systolic BP out of which two have high pulse pressure.

Percentage of Smokers – of the 53 patients selected, 30.18% (16/53) were bidi/cigarette. Smoker (1-2 bundle/day for at least 10-12 yrs.) and 11.31% (6/53) were tobacco chewer. Studies conducted on Indian coronary artery disease patients who are settled in united state found that smoking is a less commoner risk factor for coronary artery disease in patients of Indian origin compared to whites.

Studies conducted on Indian population showed that the consumption of tobacco is 6.1% of the worlds total un-manufactured tobacco, 20% is in the form of cigarettes, 40% is in the form at bidies and rest as smokeless tobacco products. Studies have shown that 40-50% at the males in India are smoker. Smoking increases the risk of coronary artery disease 3-5 times.

In our study smoking as a risk factor was present in 30.18% (16/53). In present study group there were no female having smoking habit probably this is due to relatively low figure of female patients in our study group. It also shows low incidence of smoking habit in females of Bundelkhand region.

A comparative study of smoking[71] found out incidence of smoking to be 35.8% in urban male and 1.4% for urban female in North India. Corresponding figure for smoking for rural males and females was 54.7% and 34.3% respectively in the same study. In present study 11.31% (6/53) were having tobacco chewing habit. Out of them 9.43% (5/53) were male & 1.88% (1/53) was female. In our study 11.32% having alcohol as a coronary risk factor.

A study of ***H.S. Rissam et al*** shows that prevalence of diabetes mellitus in coronary artery disease is about 20% patients and

additional 20% may be having impaired glucose tolerance even moderate elevation of glucose in Indian is associated with increased risk of coronary artery disease while in our study 16.98% were diabetic for at least 5-10 years.

Masaharre, Ishihare MD, Ph D et al, Hiroshima Japan, 199 were studied 611 patients of Acute Myocardial Infarction, 20% (121/611) patients were having NIDDM. In our study diabetes is present in 16.98% (9/53) patient.

In previous studies mortality after Acute Myocardial Infarction was 50% greater in diabetic patients as compared with non diabetic patients. In our study group mortality in diabetic was about 5.66% (3/53). While in non diabetic group it was 7.55% (4/53).

In our study the average total cholesterol of patients was 208.88. Maximum patients 47.17% (25/53) were having total cholesterol value > 240 mg/dl and 2nd highest group of patients were having total cholesterol level > 220 mg/dl.

The range of total cholesterol seen was 156-266 mg%. ***Krishnaswamy et al*** in his study of lipid profile of 877 CAD patients found mean total cholesterol in CAD patients to be 209.54 compared to 208.88 in our study.

A study by ***Gupta R, Kaul V, Prakash H, Sarna M, Singhal S, Gupta***[72] UP at Monilek Hospital and research Centre, Jaipur in 200 of lipid abnormalities in coronary heart disease patients found that levels of total cholesterol was not significantly higher in coronary artery disease patients compared to healthy population who were

not having Acute Myocardial Infarction. In present study 88.67% (47/53) patients having total cholesterol level significantly higher i.e. > 200 mg/dl the cause behind this may be that these patients had hypertension and diabetes as associated coronary risk factor.

In the present study 53 patients of Acute Myocardial Infarction were taken. Chest pain was a common & predominant symptom reported by the subjects in the present study i.e. 79.25% (42/53). In remaining number of patients chest pain was absent but main symptoms were nausea, vomiting, palpitation, sense of impending doom or hemiplegia/hemiparesis and on ECG recording Acute Myocardial Infarction was diagnosed.

In our study group maximum patients 33.96% (18/53) belonged to antero-lateral Myocardial Infarction group while 16.98% (9/53) were having extensive anterior wall Myocardial Infarction. Antero septal Myocardial Infarction was present in 15.09% patients & 16.98% presented as having extensive anterior & inferior wall Myocardial Infarction. Inferior wall Myocardial Infarction and inferopost wall Myocardial Infarction present in each 5.66% patient. In this study 3.7% patients had no ECG abnormality but they were Trop T +ve cases.

The study by ***M Schofield et al (1986)*** shows that in patient of coronary artery disease the evidence of isolated systolic dysfunction was 25%.

The study done by ***SH Poulsen et al[17] 1997***. This study was done on group of 63 patients of Acute Myocardial Infarction and showed that the evidence of ventricular dysfunction was present in 83% (53/63). Out of them 21% (13/63) had isolated systolic

dysfunction, 24% (15/63) had isolated diastolic dysfunction, while 38% (24/63) showed both systolic as well as diastolic dysfunction and 17% (11/63) were showing no systolic or diastolic dysfunction. Another study which is also preformed by ***SH Poulsen et al in 2001***, on 183 consecutive patients of acute Myocardial Infarction Preserved left ventricular systolic & diastolic function was present in 39.89% (73/183), Isolated left ventricle systolic dysfunction was present in 5.45% (10/183), left ventricle diastolic dysfunction with preserved systolic function was present in 32.79% (60/183) and combined left ventricle systolic & diastolic dysfunction was present in 21.86% (40/183).

In our study isolated systolic dysfunction was present in 3.77% (2/53) and isolated diastolic dysfunction was present in 22.64% (12/53) while 39.63% (21/53) developed both systolic and diastolic dysfunction. The result of our study in almost same as the study performed by ***SH Poulsen 2001***, in our study percentage of systolic & diastolic dysfunction is higher in comparison to this study while the incidence of isolated diastolic dysfunction was more in Poulsen's study as compared to my study. The presence of left ventricle diastolic dysfunction with preserved systolic dysfunction is associated with increased morbidity & morality.

AV Goldersis M et al (1992) in their study showed that in coronary artery disease, evidence of reduced fractional shorting in men & women was 18.9% & 10.9% respectively.

Gheorghide M et al[19] 1988 reported that cardiogenic shock occurred in 6% - 20% of patients with Acute Myocardial Infarction. Cardiogenic shock occurs when more than 40% left ventricle get damaged by infarction. Cardiogenic shock occurs in 80% due to

damage of left ventricle & 20% due to ventricular septum defect + mitral regurgitation. Recent large randomized trials of thrombolytic therapy and observational data basis report an incidence rate in the range of 7% about 10% of patients with cardiogenic shock present with this condition at the time of admission whereas 90% develop it during hospitalisation (This low output state is characterized by elevated ventricular filling pressure, low cardiac output, systemic hypotension & evidence of vital organ hypoperfusion). The course of this would more likely be old age, history of a prior Myocardial Infarction, or congestive heart failure, and to have sustained an anterior infarction at the time of development of shock. When shock develops in the course of Acute Myocardial Infarction, it usually due to infarct extension. Mortality rates of about 70 percent. In our study cardiogenic shock was present in 20% (11/53) patients. 63.64% patients were presented with cardiogenic shock (7/11). While 36.36% (4/11) were developed it during hospital stay. The incidence of cardiogenic shock was high in our study as compared to above study the reason may be the ignorance of chest pain due to confusion of myocardial infarction pain, to pain due to gastritis. Most of the time patients were ignored chest pain as they thought that it was due to indigestion or other gastrointestinal trouble. Another cause is large percentage of antero-lateral and extensive anterior wall Myocardial Infarction in our study group. Mortality rate due to cardiogenic shock in our study was 18.18% (2/11) which is very low in compared to above study possibly because of small group of patients in our study. Another reason may be that because of ignorance, some patients had died before reaching the hospital.

In the Worcester heart attach study over a period of thirteen years (from 1975 to 1988) the incidence of cardiogenic shock ranged from 6.7% to 9.1% in a sample of 4762 patients with a mortality rate

of 74%-82%. In comparison our study shows incidence of cardiogenic shock 20% and morality was 18.18%. This is may be due to small sample size of our study group and due to shorter duration of study time. This also shows large incidence of cardiogenic shock in Bundelkhand region.

Bengtson et al[23] showed that most important independent predictor of in hospital and long term mortality rates was patency of the infarct related artery which we could not assess in our study group due to unavailability of angiography study facilities in our department.

In present study cardiogenic shock was present in 20% of Acute Myocardial Infarction. In our study maximum incidence was present in (40-49yrs) and (60-69yrs) age group. Cardiogenic shock was usually present in those who had multiple risk factors predominantly, diabetes mellitus, hypertension or increased serum cholesterol level and they were also having associated moderate to severe ventricular dysfunction i.e. ejection fraction below 40%.

Right ventricular infarction has been frequently accompanied in inferior left ventricular infarction or rarely occurs in isolated form. Various pathological studies reveal that right ventricular infarction is present in 14% to 34% of patients with transmural left ventricular infarction. In approximately 30% of the patients (20% - 45%) with inferior left ventricular wall myocardial infarction there is some evidence of right ventricular necrosis. In present study right ventricular infarction present in 3.77% (2/53). One patient had infero-posterior Myocardial Infarction+Right Ventricular Infarction the other one had infero-posterior Myocardial Infarction + Right Ventricular Infarction along with ant wall Myocardial Infarction. Smoking was the common risk factor in both and one patient had

serum cholesterol level > 200mg/dl out of the two patients one had died who was having systolic as well as diastolic dysfunction and belonged to age group 60-69 years.

Mechanical causes of congestive heart failure following myocardial infarction includes ventricular septal defect, mitral regurgitation & free wall rupture. As a group they are responsible for 15% mortality after Myocardial Infarction.

The incidence of rupture of the interventricular septum is probably in the range of 2% of Acute Myocardial Infarction patients Biventricular failure generally ensures within hours to day.

Di Suname et al 1997 had reviewed 34 patients of Acute Myocardial Infarction out of them 1-2% represented ventricular septal defect as a serious complication. In our study 1.88% patients presented ventricular septal defect as a mechanical complication of Acute Myocardial Infarction. The result of our study is the same as above.

Cooley DA[40], 1998 reported that the post infarction ventricular septal rupture is an uncommon but serious complication of Acute Myocardial Infarction.

Mesa Garcia JM et al[36] 1998 reported that in Acute Myocardial Infarction patients the most frequent mechanical complication is ventricular rupture which is the cause of death in 26% of cases of Acute Myocardial Infarction. In present study the mortality is 100% in the cases of ventricular septal defect. But ventricular septum defect was not the sole cause of death but there were other associated complications, which had led to mortality of the patients of this group. As on echocardiographic examination there is large defect in ventricular septum due to massive infarction.

Partial or total rupture of a papillary muscle is rare but often fatal complication of transmural myocardial infarction.

Honma et al*[43] *1997, Tokyo Japan evaluated 223 patients of Acute Myocardial Infarction, mitral regurgitation was present in 21% of the patients at the onset & developed in 18% of the patients during followup. ***Benico Barzilia et al*** in their study documented that 43% of the total patients were having mitral regurgitation. In present study the incidence of mitral regurgitation was 7.58% (4/53).

Free wall rupture of the infarcted ventricle occurs in upto 10% of the patients due to thinness of the apical wall and usually leads to hemopericardium. It is a very rare finding. ***Tikiz H et al***[38] had done study on 350 patients of Acute Myocardial Infarction. The over all incidence of left ventricular aneurysm was 11.7% (41/300). In our study only one patient had developed left ventricular aneurysm i.e. 1.88% (1/53). The patient was having extensive anterior wall myocardial infarction along with inferior wall myocardial infarction Obesity and increased S. Cholesterol level (>200 mg/dl) were the risk factors.

Arrhythmias following Myocardial Infarction can be ventricular or atrial. Prevalence of non-sustained ventricular tachycardia varies in relation to timing of myocardial infarction. Non-sustained paroxysms of monomorphic or polymorphic ventricular tachycardia may be seen in up to 67% of patients. These non sustained ventricular tachycardia do not appear to be associated with an increased mortality risk either during hospitalization or over the first year. Episodes of sustained ventricular tachycardia during the first 48 hours following Acute Myocardial Infarction are associated with hospital mortality of about 20 percent. Now the incidence of ventricular fibrillation following myocardial infarction <10% out of

which 60% developed within 4 hrs. of myocardial infarction while 80% within 12 hours of the onset of symptoms.

Bobrov VA et al[58] studied on a group of 85 patients of acute myocardial infarction admitted within 12 hrs. of the myocardial infarction Ventricular tachycardia developed in 50% of the patients with acute large size infarct. In present study ventricular arrhythmias present in 3.77% (2/53) which was 28.57% (2/7) of total arrhythmias developed in our study group.

Eldar M et al[60] ***1998*** had observed that paroxysmal atrial fibrillation was a frequent complication of Acute Myocardial Infarction. The incidence was (8.9% 9.9%) at the time of admission. While in present study the incidence of atrial arrhythmias was 9.43% (5/53) which is 71.43% of total arrhythmias developed in study group. Incidence of total arrhythmias developed following myocardial infarction was 13.21% (7/53).

25% to 40% of patients with Acute Myocardial Infarction have electro cardiographic evidence of sinus bradycardia within the first hour of the onset of symptoms.

A-V block present in patients of Acute Myocardial Infarction due to block on the following level eg A-V node, intraventricular node and bundle of His. In our study group A-V block was present in 9.4% (5/53) patients. In various studies first degree A-V block was present in < 15% patients, 2nd degree in upto 10 percent of patients of Acute Myocardial Infarction while complete AV block develops in 5-15% of patients. The incidence may be even higher in patients with right ventricular infarction.

Altun A et al[62]***, 1988*** reported that A-V block is a frequent complication of inf wall Myocardial Infarction, study done by

Kosuzue M et al 2001 found out the same results. In our study out of 5 patients of A-V block, 3 patients were having inferior wall Myocardial Infarction i.e. 60% (3/5).

The incidence of pericardial effusion following Acute Myocardial Infarction is approximately 25% of patients. In present study pericardial effusion developed in 18.86% (10/53) of patients following Acute Myocardial Infarction. Out of 10 patients of pericardial effusion one had died because of massive pericardial effusion. In our study group pericarditis (alone) is present in 1.88% (1/53) of patient.

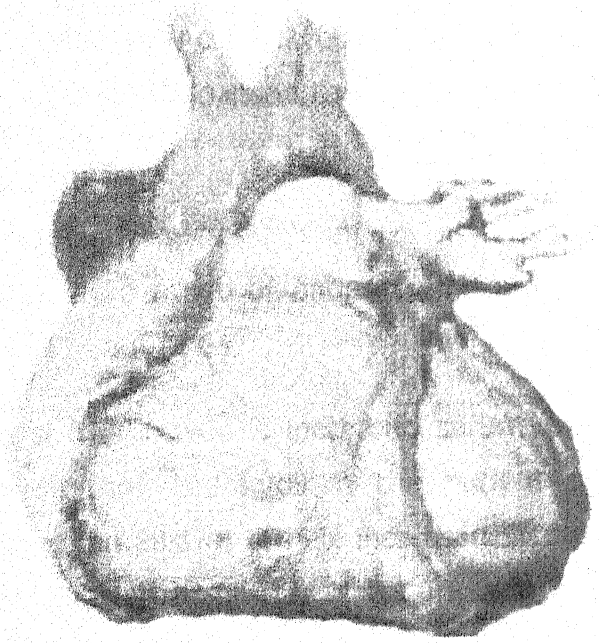
Recurrent chest pain after Acute Myocardial Infarction is another common complication of Acute Myocardial Infarction. The incidence of post infarction angina without reinfarction is between 20% and 30%. This complication may occur due to recurrent infarction the incidence of this complication of Acute Myocardial Infarction is 5%-20% within the first 6 weeks and may be somewhat higher in patients who have received thrombolytic therapy. In our study recurrent chest pain was present in 3.77% (2/53) patients ***Marmor*** reported that recurrent infarction occurs more in obese, females & patient with non transmural myocardial infarction, & patients with diabetes mellitus those with a previous myocardial infarction. In present study both patients were male this is may be because of low percentage of female patients in our study group both were obese and one had increased serum cholesterol level.

Cerebrovascular accident as a complication of Acute Myocardial Infarction occur in < 10% of cases. Most important contributor of this is left ventricular mural thrombus. In our study this complication was present in 3.77% (2/53) both patient had CT-scan proved large sized infarction. Both were having ventricular

fibrillation and both were got deteriorated & died in hospital. Both were having extensive anterior wall Myocardial Infarction on ECG.

In our study 7.54% (4/53) patients had developed no complications.

About 50% of the deaths associated with acute myocardial infarction occur within 1 hour of the event. In present study of group of 53 patients of Acute Myocardial Infarction the total mortality was 13.21% (7/53). Mortality in our study group maximally contributed by ventricular dysfunction. Though single mortality was contributed by more than one complication.



CONCLUSION

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Following conclusions can be derived from our study

(1) Incidence of Acute Myocardial Infarction is more in male patients in comparison to female patients (Male = 41, Female = 12)

(2) Patients of myocardial infarction has average age of 55.32 years. Percentage of patients who are below 50 years. is 14 (26.42%) out of 53.

(3) Major risk factor is increased serum total cholesterol level (more than 200mg/dl) in the patient of Acute Myocardial Infarction 88.67% (47/53). Increased serum total cholesterol is individually major risk factor in both male & female group (Male = 71.69%, Female = 16.98%).

(4) Hypertension is the second major contributory risk factor in the coronary artery disease (35.84%) in both male & female patient (32.07% & 3.77% respectively).

(5) Smoking is the 3rd most common risk factor in study group in whole as well as in Males.

(6) Chest pain is the most common presenting symptom in the coronary artery disease.

(7) Rural, Urban division of the patients was as follows in our study

* Rural background - 43.40% (23/53)
* Urban background - 56.60% (30/53)

(8) Ventricular dysfunction is major complication following Acute Myocardial Infarction. (66% = 35/53).

(9). In large number of patients systolic as well as diastolic both dysfunctions present. (39.63% - 21/53).

(10) Only diastolic dysfunction was present in 22.64% patients.

(11) 33.96% patients did not show any regional wall motion abnormality following Acute Myocardial Infarction. Their 2D-echocardiographic & M-Mode study is within normal limit.

(12) Isolated systolic dysfunction was present in only few patients i.e. only in 3.77%. So isolated systolic dysfunction is less common following Acute Myocardial Infarction.

(13) Cardiogenic shock is the second most common complication following Acute Myocardial Infarction. It is present in 20% of patients.

(14) Pericardial effusion is also the major complication following Acute Myocardial Infarction in our study group (18.86%) while pericarditis was present in single patient. This probably was reflection of heart failure.

(15) Thromboembolic episodes are present in 2 patients & mortality is 100% following this.

(16) Arrhythmias are present in 13.2%. Out of which ventricular arrhythmias are more common and fatal in comparison to atrial arrhythmias.

(17) In our study right ventricular infarct was present in 2 patients, ventricular septum defect and left ventricular aneurysm were each present in one patient each, while mitral regurgitation due to papillary muscle rupture was found in 4 patients.

(18) Recurrent angina was reported in 3.77% patient (2/53).

(19) 7.54% patients showed no complication following Acute Myocardial Infarction.

(20) Total mortality is 13.2% (7/53) mainly due to cardiogenic shock.

(21) A-V block was detected in 9.4% patients of our study group.

Observations of the present study are in conformity with other studies.

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